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THE EVOLUTION AND DEVELOPMENT OF SURGICAL INSTRUMENTS

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VI. HEAD-SAWS

THE use of the head- or hand-saw for opening up the skull goes back at least four thousand years, and its employment in the near East about that period is proved by a skull excavated by the late Mr. Starky at Lalash a short time ago. This discovery is of special interest as it shows that the operation was carried out in the same manner as that employed by the ancient people of Peru, the cranium being first sawed in two directions and a square piece of bone then extracted.

As in the case of other early instruments used for surgical purposes, the primitive flint implement then employed was succeeded later on by small saws with blades of copper or iron.

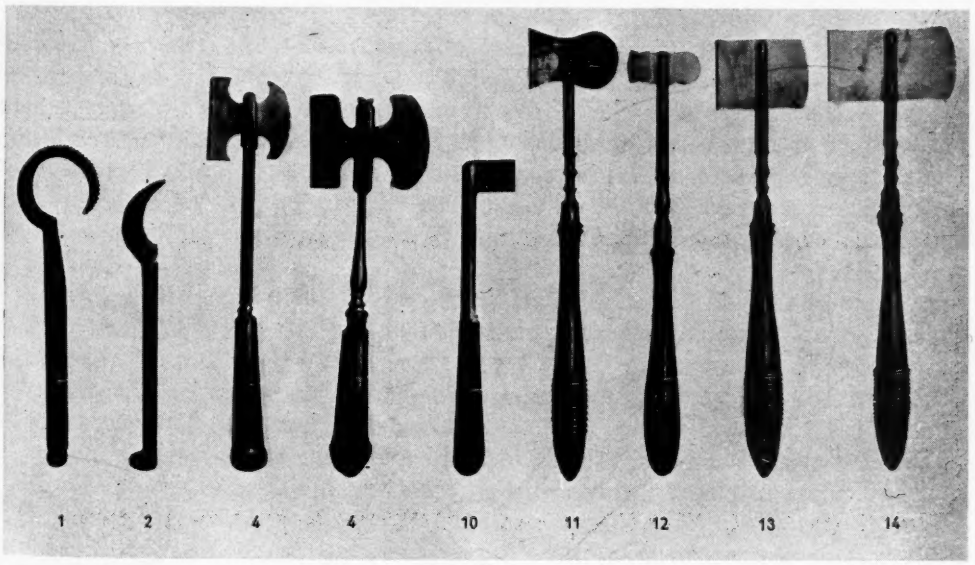
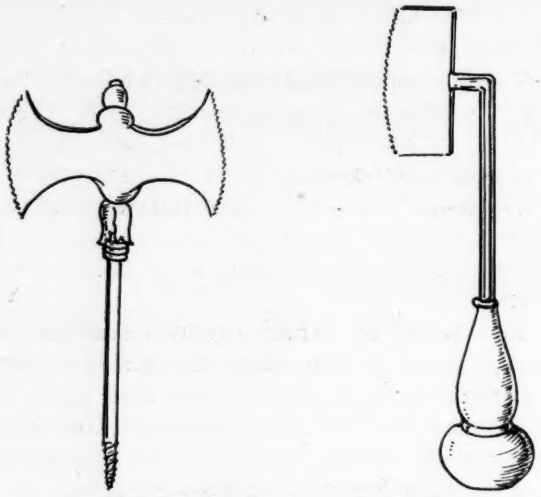


FIG. 298.—Head-saws: 1, Albucasis (twelfth and thirteenth centuries); 2, Wryghtson (fourteenth century); 4, Andreas della Croce (sixteenth century); 10, Cockell's (1783); 11–14, Hey's (1803).

The first representation we have of the head-saw is that given in the *Chirurgie* of Albucasis in manuscripts of the twelfth and thirteenth centuries, where it is depicted with a slightly curved blade and also with a blade almost circular in shape (*Fig.* 298, 1).

He describes the instrument as "the little saw sometimes used to cut away the spaces between the holes made in the skull by the trepan".

In the fourteenth century there is evidence from the drawing in Wryghtson's manuscript at St. John's College, Oxford, that a crescent-shaped blade with



FIGS. 299, 300.—Head-saws as used by Andreas della Croce (1573).

FIG. 301.—Head-saw used by Ambroise Paré (1560).

FIG. 302.—The type of head-saw used by Fabricius Aquapendente (1537–1619).

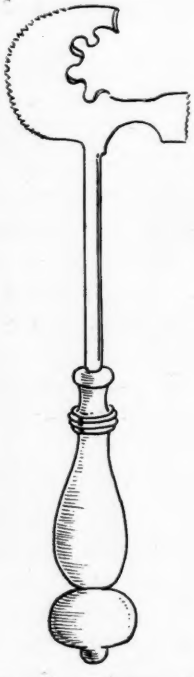


FIG. 303.—A type illustrated by Fabricius Hildanus (1560–1624).

FIG. 304.—The small hand-saw used by Scultetus (1674).

the serrated edge on the outside was used in that period (*Fig.* 298, 2), and this type probably continued in use until the sixteenth century, when we find eight different varieties of the instrument in the works of Andreas della Croce. One of these has a small oblong blade with a slightly curved sawing edge fixed to a handle, by means of which it was operated. Another has the blade curved backwards with the serrated edge on the outside, while a third is shaped like a halberd, with double saws fixed on either side of the shaft (*Figs.* 298, 4, 299).

The development from the curved into the semicircular edge seems to be traceable from the time of Wryghtson to della Croce, who shows a type with a highly convex sawing edge (*Fig.* 300). Ambroise Paré in his *Chirurgie* (1560) depicts several types of head-saw, which he describes as " saws fit to divide the skull " (*Fig.* 301), while Fabricius Aquapendente (1537–1619) figures a head-saw the blade of which is curved slightly backwards, with the cutting edge on the outside (*Fig.* 302). Fabricius Hildanus about the same period (1560–1624) gives a representation of an instrument with a head of peculiar shape (*Fig.* 303). The serrated edge is on the outside of a semicircular blade, while on the other side the curve has indentations of various shapes, probably intended for breaking away pieces of bone, and below these a small straight saw.

In 1674 Scultetus figured a small hand-saw, which closely resembles some of the later types, with which, he says, " some men cut away the distances between the holes made in the skull with the trepans and abolish rifts like hairs that do not penetrate and scrape away the rotteness of the cranium ", thus repeating the use of the instrument given by Albucasis (*Fig.* 304).

From this period the head-saw appears to have fallen into disuse for over a century, until in 1783 it was revived by Cockell, a surgeon of Pontefract, who is said to have " used the saw he devised with great advantage in extensive fractures of the skull ". The blades of Cockell's saws varied in shape, some having a semicircular sawing edge and others a smaller straight blade with convex edges (*Fig.* 298, 10).

Twenty years later, in 1803, William Hey, F.R.S., senior surgeon to the General Infirmary at Leeds, again advocated the use of the head-saw in cranial operations. It is evident from the difference in the shape of the blades that Hey gave very careful thought to the designing of his saw, as shown in the six examples which he used himself that are now in the collection of instruments in the Museum of the Royal College of Surgeons. In his *Practical Observations on Surgery* (1803), in referring to his saw, he says: " Such a saw I can with confidence recommend after a trial of twenty years, during which time I have rarely used the trephine in fractures of the skull.

" It was first shown me by Mr. (now Dr.) Cockell, an ingenious practitioner of Pontefract, to whom the public is indebted for the discovery or revival of this excellent instrument.

" Dr. Cockell's saw has a semi-lunar edge, but the edge may be straight or of any degree of convexity which may be thought most useful. The straight-edged saw executes its task with greater readiness, but the convex edge is necessary when the bone is to be sawed in a curvilinear direction. It is also useful when the thickness of that part of the cranium which is to be sawed through is very unequal."

As Cockell and Hey advocated varying degrees of convexity, it is not surprising that such variations are displayed in Hey's own set which he used in the Leeds Infirmary (*Figs.* 298, 11–14, 305, 15, 16).

Hey's original pattern had a metal stem on a wooden handle: the stem was split at its free end and the single straight blade was fitted at right angles into the slot thus formed by two screws. Of the other types he devised one had an asymmetrical semicircular cutting edge (*Fig.* 305, 18), another a single straight blade with a curved stem, while to another a projecting spring guard was added, the two pieces of steel being probably designed for cleaning the groove made by the saw (*Fig.* 298, 19–22). In 1878 Bigger described a head-saw of Hey's type which had teeth also on the concave side, so that both the convex and concave sides of the cutting blade could be employed.

In connection with the head-saws, mention must be made of the circular types introduced in the early nineteenth century.

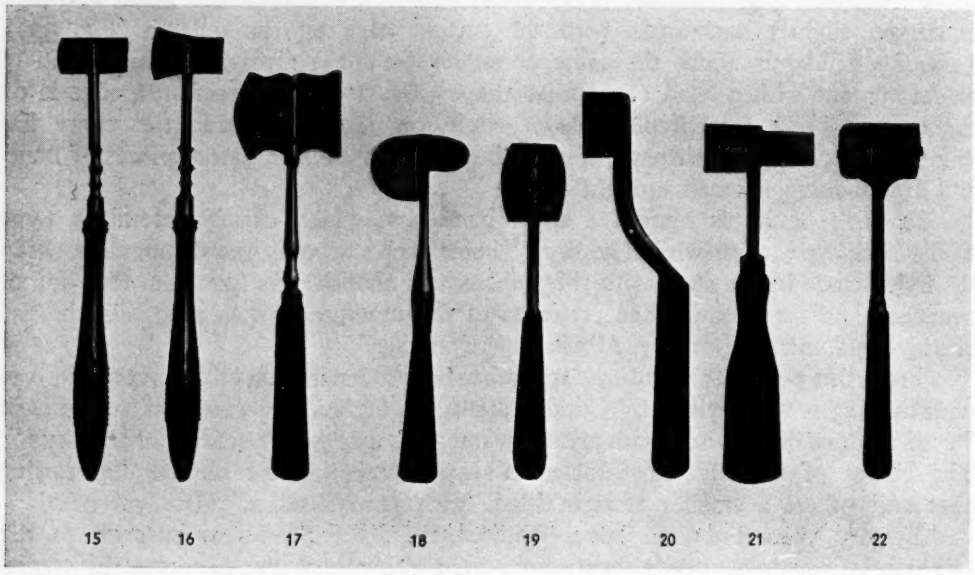


FIG. 305.—Hey's saws: 15, 16, Later patterns; 17, An early pattern; 18, With asymmetrical semicircular sawing edges; 19, With symmetrical convex sawing edges; 20, With single straight blade and curved stem; 21, With asymmetrical straight sawing edges; 22, With spring guard.

Among the earlier instruments employed in trepanation the 'cutting compasses' figured and described by Ambroise Paré in 1564 are noteworthy, as the principle was revived in much later instruments. Concerning it Paré says, "For what cause soever we cannot make use of a trepan we may employ the 'cutting compasses' to cut forth the skull. If so be much of the bone is bared as is needful, it is made in form of a pair of compasses and by means of a screw may be opened more or less as you please". With it a perforated metal plate was used to protect the cranium into which the pointed shaft of the instrument was to be placed (*Fig.* 306).

A circular saw for cranial surgery was invented by Machell in 1808, and of this he devised several types (*Fig.* 307). In introducing it he says, "the annular saw is acknowledged to supersede the use of the trephine, trepan, Hey's saws, bone-nippers, rasp, and the mallet and chisel in all cases where any portions of carious bones demands the interference of our art to remove it". He claimed it to be specially useful for clearing the ends of the ununited fractured bones. Later,

he made various modifications in his original design, and the instrument is figured in *Surgical Essays by Astley Cooper and Benjamin Travers* as " Machell's saw ".

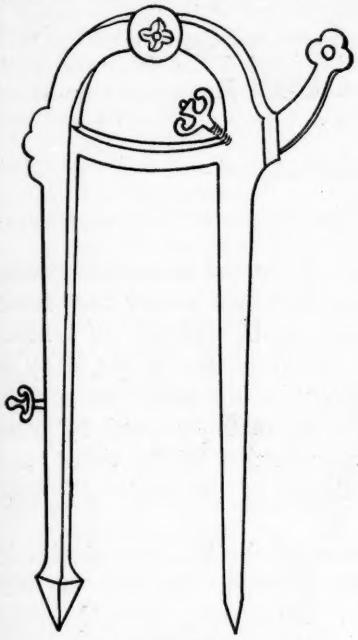


FIG. 306.—Ambroise Paré's cutting compasses (1564).

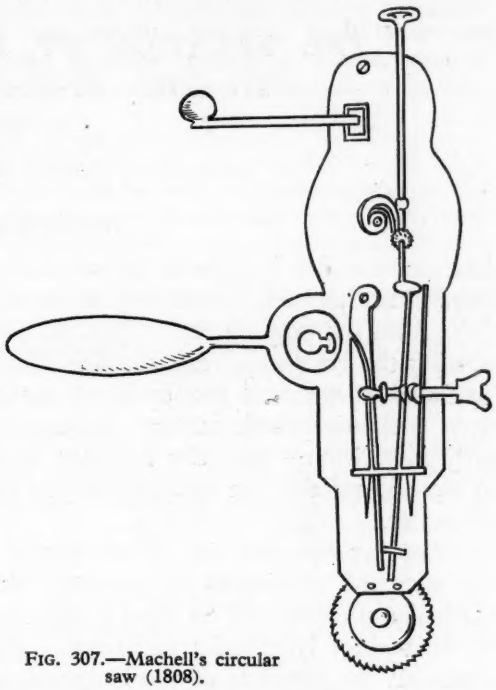


FIG. 307.—Machell's circular saw (1808).

Machell's invention was followed by the circular saw devised by Charles Griffiths, an Army surgeon, in 1815, and in conjunction with it a saw and bone-nipper which he suggested might be used in cranial surgery.

Another mechanical saw was introduced by Thal in 1823 (*Fig.* 308). In this instrument the blades consisted of circles or sections of circles of various sizes, which could be fitted to a shaft which was rotated with one hand while the other supported the whole instrument by a handle in which was mounted the main bearing through which the shaft passed.

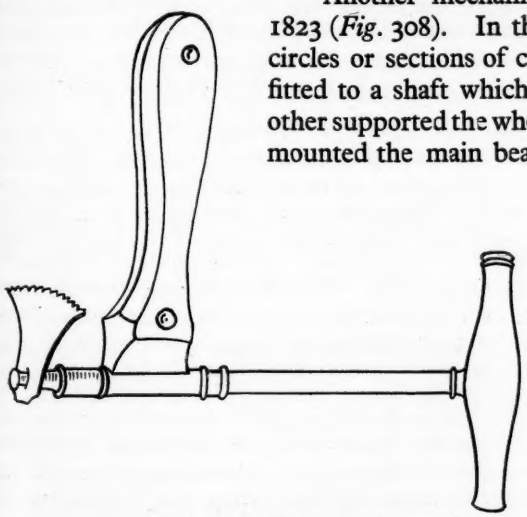


FIG. 308.—Thal's mechanical saw (1823).

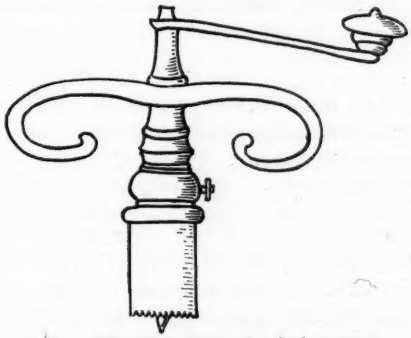


FIG. 309.—Brun's mechanical trepan.

About 1830, Brun devised his mechanical trepan which was operated with a transverse rotary handle like a mill (*Fig.* 309). This, however, like other contrivances failed to find favour with the surgeons of the time, and it gradually fell out of use.

THE RELATION OF LYMPHATIC VESSELS TO THE SPREAD OF CANCER

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INTRODUCTION

ALTHOUGH it has long been known that cancer tends to spread to lymph-glands, there is considerable uncertainty as to the exact mode in which cancer cells reach them from the primary site. This has been due chiefly to the difficulty of demonstrating the lymphatic vessels. The illustrations of the lymphatics of the body to be found in standard text-books of anatomy have been for the most part copied from such nineteenth century workers as Sappey. The methods used by these early investigators generally required an advanced putrefaction of the tissue, and so were unsuitable for research on the histological details of the mode of spread of cancer.

Another cause of the uncertainty is that accounts of the skin lymphatics in the standard text-books of anatomy are vague and obscure. Because of this, particular attention will be given in the present paper to the anatomy and physiology of the smaller lymphatics in so far as they form an essential preliminary to any attack on the problem of the lymphatic spread of cancer.

METHOD OF INVESTIGATION

It was felt that for the elucidation of the problem a reliable and easy method of injecting the lymphatics was the first essential, in order that in the histological analysis of malignant tissue any vessels which might be lymphatics could be traced by direct continuity into vessels the identification of which would be beyond all question. For this purpose it was suggested by Professor H. H. Woollard that the colloidal solution known commercially as 'thorotrast' would be a suitable injection material.

For injection of the lymphatics of the skin with thorotrast an ordinary hypodermic syringe with ordinary fine bore needle is used (it is not necessary to use the finest bore obtainable), and an intradermal injection of ½ to 1 c.c. of thorotrast is made into the skin area the lymphatics of which it is wished to fill. Warm skin used immediately after removal at operation, or skin removed at post-mortem and subsequently heated in a bath to body temperature is suitable; but whatever tissue is used, it must not be touched by fixatives before injection, and it must be as fresh as possible. The injected area is then fairly vigorously massaged for about five minutes, particularly in the direction in which the lymphatic trunks are known to run. If a radiograph of the tissue is then made, it will be found that the fine lines of the lymphatics filled with the radio-opaque thorotrast can be seen casting characteristic, dense, well-defined shadows. If the tissue be then fixed and dehydrated by the Spalteholtz method (benzene for clearing, and methyl salicylate for preserving—bleaching is generally not necessary), it will be found that the alcohol used for dehydration flocculates the colloidal thorium into opaque white masses, which retain this appearance when the surrounding tissue is made transparent by the benzene. If longer times and several changes of fluid in the dehydration and clearing stages are allowed, perfectly satisfactory specimens from very large pieces of tissue such as a whole breast can be made.

The specimen after treatment by the Spalteholtz method can be divided into blocks for thin paraffin sections or for celloidin sections without any difficulty, and when this is done

and the sections stained, the thorium can be readily recognized by indirect illumination under the microscope, or by its rather negative affinity for most stains. Using oblique illumination one sees the patches of thorium as brilliantly lit-up areas on a dark background. With stains it is found that the thorium has some affinity for both hæmatoxylin and eosin, so that somewhat amorphous, granular, or almost globular, violet-coloured masses are seen. Sometimes the processes of dehydration, clearing, and staining leave the thorotrast as hyaline masses in which small granules or globules are not very evident. In these latter cases indirect illumination leaves no doubt as to the identity of thorotrast.

Perhaps the most important technique for the investigation of lymphatic vessels in tissues which have been injected consists in the making of rather thick sections of the order of 250 to 300 μ. For this purpose it was found best to use a celloidin microtome, although where it is not necessary to have good serial sections they can be cut by hand at this order of thickness.

It was felt that at least in some cases it would be advisable to make double injections—of the blood-vessels in relation to a tumour as well as of the lymphatics. The most satisfactory way of doing this is to use indian ink and gelatin mixtures for the blood-vessels and thorotrast for the lymphatics. In this way it is possible to identify unequivocally every space and vessel in the tissue. Indian ink and gelatin mixtures pass along the blood-capillaries into the veins very readily. The injection into the blood-vessels for this part of the work was made by inserting a cannula into the main artery (of the limb, for instance), while the injection of the lymphatics was of course a filling of the vessels from the periphery.

Control investigations, as well as observations of innumerable sections, have shown that thorotrast when injected as described above into the lymphatics practically never enters the local blood-vessels. Thorotrast is likely to enter the blood-vessels only in those cases where the tissue is exceedingly vascular, with many thin-walled dilated blood-vessels, or where the blood-vessels are abnormally permeable and dilated, as in acute inflammation. In these instances a few granules of thorotrast may enter some of the blood-vessels, but it is usually easy to recognize such vessels because of their large red blood-cell content. When their connexions are traced, as can be readily done in serial sections of injected material, they are found to be branches of undoubted blood-vessels.

To make injections of the lymphatics in a part of the bowel it is necessary to insert the needle into the mucous membrane where the plexus of finest lymphatic capillaries is to be found. Usually no success is to be obtained by injecting into the muscular layers of the bowel.

STRUCTURAL BASIS FOR LYMPHATIC SPREAD OF CANCER

CRITERIA FOR IDENTIFYING LYMPHATIC VESSELS

1. A supposed lymphatic vessel, if it is a capillary lymphatic, must have a proper wall of true and complete endothelial cells only. If it is a collecting trunk its wall must consist of endothelium with surrounding smooth muscle and adventitial layers arranged in a somewhat but not altogether characteristic manner.

2. The vessel must possess valve folds projecting into the lumen if it is a lymphatic beyond the capillary stage. In its appearance the lymphatic valve does not seem to be formed by a number of separate cusps as occurs in other vessels. The valve gives the impression when seen in its entirety of consisting of a single relatively long tubular prolongation of the whole endothelial lining of a segment of a lymphatic forwards in the direction of lymph-flow into an expanded end of the next segment of the vessel. But further analysis has shown that actually there are always two semilunar cusps in exact opposition. Such valves are present at very frequent intervals, the actual distance separating lymphatic valves being much less than that in the veins of an adult. Also they are very constantly to be found at the point of meeting of two non-capillary lymphatics. The absolute distance separating lymphatic valves seems to vary with the size of the vessel, being correspondingly shorter in the smaller trunks than in the larger. The only instances where valves are normally few or absent from lymphatics are in vessels of the

parenchyma of certain organs such as the thyroid and lungs. The valves thus constitute a very important feature in the identification of lymphatics.

3. The vessel must be capable of being traced into direct continuity with other and easily identified lymphatic vessels or with lymph-glands, and in all cases ultimately to lymph-glands, the lumina being continuous up to and including the lymph-sinuses of the gland. Such continuity is best traced when the lumina of the vessels have been filled by an injection mass, and the tissue studied in thick and thin sections. Continuity cannot be traced with certainty without injection.

4. Lymphatic vessels and their plexuses have a much more characteristic appearance when seen in their entirety as in thick cleared sections where they have been injected with opaque or coloured material than in thin sections used in ordinary microscopical study. Hence this check should be used wherever possible, and is indeed an almost indispensable one.

5. In regard to the contents of the lumen it can be said that lymphatics normally do not contain blood, though the presence of a few red blood-cells in a vessel does not exclude its being a lymphatic. Many investigators have shown that after much exercise of a part or after vigorous massage there is an appreciable number of red blood-cells in the lymph flowing from the region. But although blood in large amounts is absent, lymphatics in prepared sections may contain coagulated lymph, and in this lymph there may be a few leucocytes, particularly lymphocytes. Coagulated lymph usually appears hyaline and homogeneous, but sometimes a fibrin network is visible in it. In most cases the cells of the lymph tend to become aggregated so that the lymphocytes appear as a clump at one end of the coagulum. Thirdly, in un-injected lymphatics the lumen may appear in section as quite empty, and this applies to both the very small and the large vessels. In such cases it becomes very important to apply all the other tests for identification of the lymphatic, for one has particularly to avoid mistaking a connective-tissue cleavage plane, or shrinkage area, or other artefact, or an empty vein, for a true lymphatic vessel. There may appear to be present a perfect endothelial lining, which is really only a line of flattened connective-tissue cells between adjacent bundles of connective-tissue fibres. Then, fourthly, the lumen may be empty and the vessel walls completely collapsed. Such vessels can hardly be recognized without the test of continuity and the aid of injections.

In this connexion also it is to be realized that the mere occurrence of a line of lymphocytes in connective tissue does not demonstrate the presence of a lymphatic vessel. In the first place, such rows of lymphocytes may occur around small blood-vessels, and secondly the resemblance to a cell-filled lymphatic may be suggested in sections by a mass of lymphocytes lying embedded in a region of connective-tissue cleavage. A line of such cells if repeated in the same position through a number of serial sections can obviously not be an intra-lymphatic cord of cells, but must be an infiltration of the connective tissue. The same remarks apply to columns of cancer cells lying in the connective tissue, and in this case the cancer cells may be surrounded by a collar of flattened fibroblasts, contributing very effectively to the illusion of a cord of cancer cells growing in the lumen of a lymphatic. The so-called stag-horn cords of leucocytes found in the dermis of the skin in some regions normally, and in others in diseased states, also belong to these arrangements or appearances which simulate lymphatic vessels. Actually many injections of lymphatics alone and of blood-vessels as well as lymphatics show that

these stag-horn appearances are not lymphatic vessels at all, but sometimes the venules, sometimes the arterioles and their blood-capillaries. It is apparent in *Figs.* 310, 311 that the lymphatics with their injected thorotrast are distinguishable

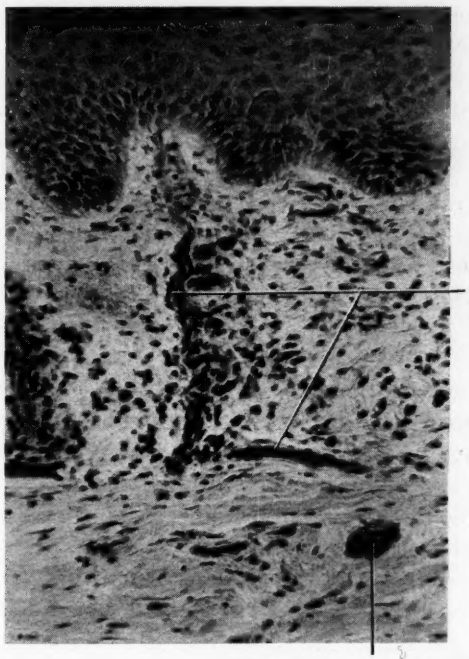

"Stag-horn" blood-vessel injected with gelatin-indian ink. Note perivascular leucocytes
Lymphatic injected with thorotrast. Note absence of perivascular leucocytes

FIG. 310.—Section of skin 1 cm. above proximal edge of epithelioma of leg (No. 50). Blood-vessels injected with diluted indian ink, lymphatics with thorotrast. Fine granules of ink can be seen in the gelatin in the "stag-horn" blood-vessels. Thorotrast in the lymphatic appears quite different. No perivascular leucocytes around the lymphatic. 15 μ section. ($\times$ 175.)

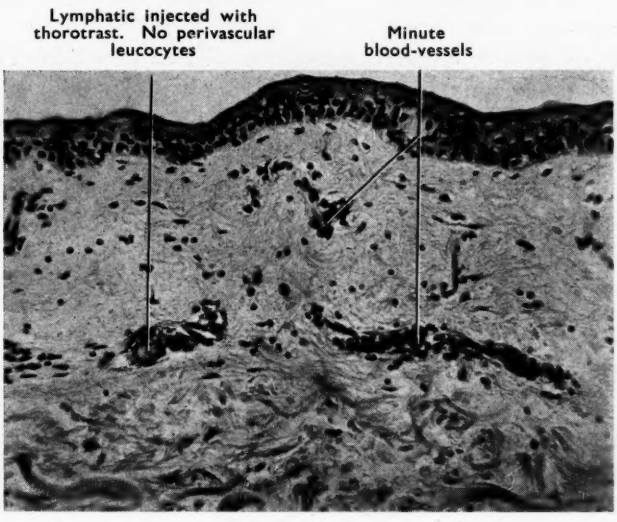


FIG. 311.—Section of skin overlying cancer of breast (No. 6). Thorotrast injection of lymphatics. Note differences from blood-vessels. 7 μ section. ($\times$ 175.)

from the stag-horn structures. It is usual in normal and in some pathological states to find the lymphatics quite devoid of any collar of surrounding lymphocytes, and thus contrasting with the blood-vessels, which may be enveloped by such a sheath of cells in certain pathological conditions.

Enough has been said to show how much caution is necessary in examining the lumen of a possible lymphatic vessel.

6. Lastly, great caution must be used in diagnosing the presence of a lymphatic in an area or region of tissue where lymphatics are not ordinarily found. Thus, most anatomists believe there are no lymphatics in bone, in the central nervous system, or in cartilage. They are not found in tendon, and they are rarely situated in the substance of deep fascia. They are rare in striated muscle, where one usually finds them only in the larger loose connective-tissue strands which convey blood-vessels to or through the muscle.

For the sure identification of lymphatics none of the above six points can be neglected.

ARRANGEMENT OF NORMAL LYMPHATICS OF HUMAN SKIN

This aspect of the subject will not be treated exhaustively in the present paper, but the main features will be summarized. The account here given is based upon the consensus of opinion of anatomists, whose writings on this subject, though not very easily accessible, are really fairly numerous. It is also based on a considerable amount of control work which has been done as part of the investigation of cancer spread and as purely anatomical work. The work on normal skin includes injections of skin lymphatics in both living and dead adult human material, fœtal skin from practically all the main regions of the surface, skin which has been affected by lymph œdema, skin of tubed pedicles, and skin from various kinds of grafts. Also double injections, of both lymphatics and blood-vessels, have been done on both adult and fœtal material. Accounts of this work have been given as papers and demonstrations at the meetings of the Anatomical Society of Great Britain and Ireland, but publication of the results has been delayed until the work has been completed.

1. The Plexus of the Superficial Part of the Dermis (*Figs.* 312–315).—This is a plexus of continuously communicating vessels joining one another by loops, and only occasionally possessing blind ends or large diverticula. It lies in the superficial part of the dermis surrounded by the compact fibro-areolar tissue of this layer, and is placed for the most part slightly more deeply than the arterioles which give off the blood-capillary loops of the dermal papillæ. Draining veins are usually deeper than these lymphatic capillaries. Their relation to the arterioles and venules is not absolutely constant. All three are very close together and a different relation from that just described is sometimes found.

The diameter of these lymphatic capillaries is generally greater than that of the corresponding blood-vessels. In this finest plexus of lymphatic capillaries of the skin the size of the lumen is fairly regular when there is no considerable distending pressure within it. In the human dermis there are rarely any great bulgings or sacculations along the course of these vessels in the normal state, and one can often trace the vessels for considerable distances without meeting any appreciable change in the size or shape of the lumen. Sacculations may occur to some extent when the vessels are distended (*see Fig.* 317).

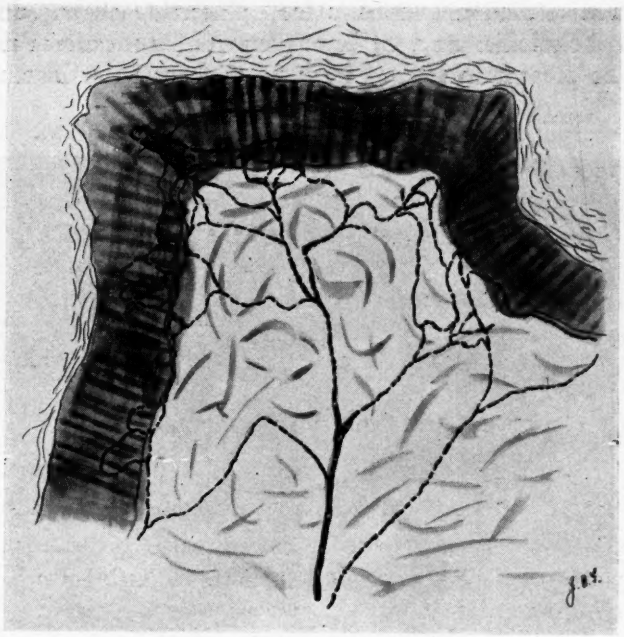


FIG. 312.—Drawing of lymphatic plexus of dermis of skin overlying a cancer of the breast (No. 6). Individual papillæ are not shown, but the plexus associated with them is seen. Thorotrast injection of lymphatics. (× 120.)

FIG. 313.—Drawing of a hand-cut section of skin from dorsum of foot of normal adult (No. 46). Lymphatics injected with thorotrast, blood-vessels with indian ink mixture. Note relatively wide-meshed plexus of lymphatics in this region. (× 75.)

It is common to find a lymphatic loop projecting slightly into the base of a papilla, and if the papillæ are very large then quite a plexiform net of lymphatic capillaries may be present in them. In some of these latter instances one or two

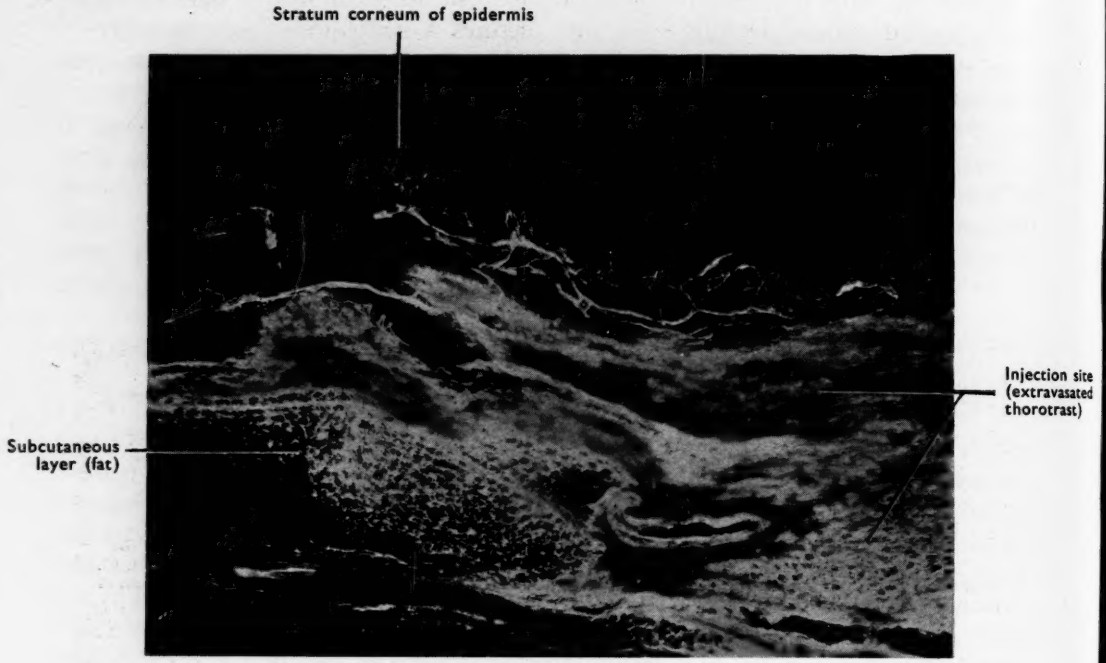


FIG. 314.—Photograph showing normal lymphatic plexus (thorotrast injected) in skin overlying a cancer of the breast (No. 6). Note the loops of papillary lymphatic plexus of capillaries. Dark-ground illumination. Section 300 μ thick. (× 10.)

blind diverticula may be found projecting from some portion of the complex lymphatic loop. Also in some regions such as the palm or the sole, frequently blind diverticulæ of lymphatics may be found projecting up into the more narrow papillæ. But in such cases it is common to find the tip of the diverticulum turned back towards the base of the papilla, as if it were in a stage of incomplete growth towards the formation of a continuous loop. In all regions there are many papillæ, particularly the smaller ones, devoid of any trace of lymphatic capillaries. One may say that the presence or absence of papillary lymphatics seems to depend upon the size of the papillæ, which in turn varies with the cutaneous region considered and is altered by many pathological conditions (*Fig.* 315).

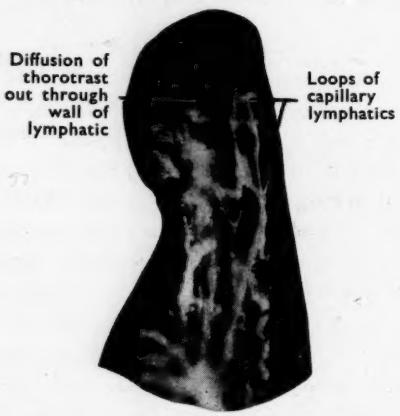


FIG. 315.—'Blocked-out' photograph under dark-ground illumination of an enlarged dermal papilla which was cut off from one of the pieces of skin shown in *Fig.* 332. Lymphatic capillaries filled with thorotrast, some of which has diffused out through the endothelium. Note the continuous network and the absence of dilatation. (× 50.)

The mesh of this most superficial plexus has some sort of uniformity of size and shape in different regions of the body, but constancy and regularity of pattern are not of a very high order. The network is in most regions

a relatively wide-spaced one. Thus on the dorsum of the foot a lymphatic capillary may be traced for more than a millimetre (in a stained slide) pursuing a fairly straight course, and without its being joined by any other lymphatic vessels during that length. This is a much longer interval than occurs in other regions such as the sole of the foot. But generally one pictures a wide-meshed plexus of fairly straight narrow vessels, the whole lying in the superficial part of the dermis in a plane parallel to that of the skin surface, and with its elements in continuity over the whole area of the body skin.

Valves have not been found in this plexus, and the anatomical evidence is quite definite that the plexus is continuous over the whole surface of the body, including the midline and the transumbilical horizontal line. The walls of the vessels of this plexus consist of endothelium alone. There is no differentiation of adventitia or formation of muscular coats around these vessels.

FIG. 316.—Drawing of same tissue as shown in *Fig.* 313. Section through deeper part of dermis. This shows a typical small lymphatic trunk with valves. Note the blood-capillary plexus on the walls of the lymphatic trunk. (× 18.)

2. The Deeper Plexus of the Dermis (*Fig.* 316).—The vessels here form a series of collecting radicals rather than a true plexus, although anastomoses are fairly numerous, and are even found to some extent across the midline of the body. But in the latter case it is probable that valvular arrangements similar to those described in the stomach[1] guide the lymph to one or other direction in such anastomosing vessels.

The more minute lymphatic capillaries are for the most part absent in this zone, although they may be seen here in relation to the various skin-secreting glands. The situation of these first trunks is in the deeper part of the dermis and passing into the subcutaneous fatty tissue. In size the vessels vary considerably. One may roughly group them into a smaller set which are truly in the dermis and have a lumen diameter (measured on the stained slide) of from two to ten times that of the lymphatic capillaries of the superficial part of the dermis; and a larger set, the lumen diameter of which may be large enough to render them visible to

the naked eye (about twenty times that of the more superficial lymphatic capillaries), and which lie for the most part at the junction of the dermis and the subcutaneous fat. In regions such as the sole of the foot and palm of the hand these valved vessels of the dermis are much more numerous. There one can make arbitrary classifications of them into several layers of plexuses, although anastomoses between such layers are numerous.

In most regions the lymphatics of this part of the dermis are joined to the superficial plexus by oblique or more or less vertical vessels which drain the fine plexus, and often appear in sections perpendicular to the skin surface like the trunks of trees (*Fig.* 312). These vertically disposed trunks may possess valves at their junction with the deep dermal plexus, and this is the first appearance of valves as one passes from the initial capillary plexus of the lymphatic system towards its glands. The collecting trunks in the deeper layer of the dermis present the typical appearance of lymphatic vessels as depicted by Poirier[2] and other writers. They possess valves spaced generally less than a millimetre apart (as measured on the stained slide), and the vessel is constricted on the distal side of the valve, and has a saccular dilatation of its lumen on the proximal side of the valve cusps. The direction of the vessels is more or less that of the flow of lymph from the particular area of skin towards the focal glands, or parallel with the direction of the subcutaneous trunks underlying the dermis. This deep dermal plexus lies in a plane more or less parallel to that of the superficial plexus, although arcade arrangement of the vessels arching up somewhat towards the skin surface is fairly often seen (*Fig.* 311).[3]

3. The Lymphatics of the Subcutaneous Tissue (*Figs.* 317, 318).—There are a few points which may be added to the common text-book descriptions of these vessels, which were very well described by authors of the previous century such as Sappey,[4] and more recently by such authorities as Poirier[2] and Rouvière.[5]

The lymphatics in this tissue are chiefly large draining trunks with very few of the smaller vessels and very few capillary lymphatics. Although these vessels tend gradually to approach the line of the main draining veins of the subcutaneous tissue, especially in the limbs, yet there is no direct or very close association. This is true particularly in the more distal parts of the limb. The relation of these lymphatics to the deep fascia needs emphasis. They lie in the superficial fascia or fat, for the most part not directly on and rarely in the deep fascia. They perforate the deep fascia at certain focal points or more strictly speaking areas, which are correctly given in the ordinary text-book. One may mention here that the perforation of the deep fascia at the axilla is spread over a relatively wide area, and perforations of the deep fascia are usually present over the flexor surfaces of the main joints such as the elbow and knee. Deep fascia itself is practically devoid of lymphatic vessels, and this fact is to be correlated with the paucity of all vessels in the deep fascia. There is no substantial capillary lymphatic bed actually in the deep fascia, nor do lymphatic trunks generally run for any appreciable distance in it. This fact of descriptive anatomy can also be correlated with the findings in work recently done by Pullinger and Florey,[6] in which they showed how the development of dense fibrous tissue causes a retrogression and ultimate disappearance of lymphatics. The relatively clear separation of the superficial or external lymphatics from the deep or internal is a matter of the first importance. The findings of the anatomists such as Sappey[4] and Poirier[2] and the confirmation of

their statements obtained in this work are quite definite, although they are negative; and they do not conflict with the clinical picture of lymph œdema, in which one may find the deep fascia acting as a perfect barrier separating œdematous superficial

FIG. 317.—Drawing of barium-injected lymphatics in the scalp of a human fœtus (No. 57). Lymphatics are distended. Surface view. (× 13.)

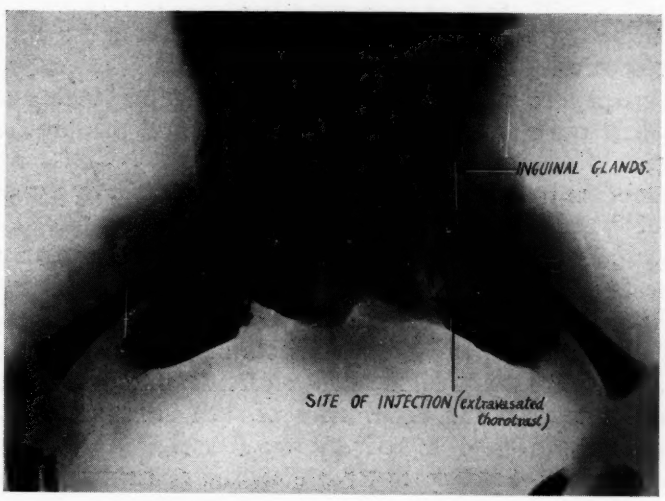


FIG. 318.—Radiograph of human fœtus No. 26. Thorotrast injections to show normal subcutaneous lymphatic trunks and inguinal glands. (× ½.)

tissues from normal deep tissues. This separation is the basis of the Kondoleon operation, the limitations of which may be due to the fact that the deep lymphatics in limbs are limited in site to the immediate neighbourhood of the chief arteries and veins, and come from such structures as the synovial membranes, but not to any important extent from the muscles or fascial septa.

There has been an opinion prevalent in the past that the membranous fascial layers of the body are riddled with lymphatics. Actually nowhere in the literature are there any convincing pictorial demonstrations of a lymphatic plexus in the deep fascia. But one finds that in spite of the fact that Sappey himself was perfectly clear about the absence of extensive anastomosis across the deep fascia, his distinction between superficial and deep lymphatics has been confused and lost in later clinical writings. There is no real evidence for such a postulate as a 'deep fascial plexus' of lymphatics; and for the purpose of historical accuracy it must be stated that the idea is quite foreign to the writings of Sappey.[4] This interpretation of Sappey's writings is confirmed by Poirier.[2]

There is one modification of the above account of the subcutaneous lymphatic trunks which requires mention. Where large blood-vessels pierce the deep fascia in order to reach some important superficial organ such as the breast, one finds a perivascular lymphatic plexus running with these blood-vessels; but exactly how far it ultimately goes towards the gland or other organ or the skin has yet to be determined. At least it is thought that anatomical anastomosis from such perivascular lymphatic plexuses adhering closely to the larger blood-vessels to the ordinary lymphatics of the skin and glands is insignificant. And on this point the findings of the anatomists are in agreement with the clinical observations on those cases of lymph œdema of the whole upper quarter of the trunk following surgical 'clearing of the axilla'.

While this paper does not require the assembling and assessing of all the evidence germane to the above account of the lymphatics of the human skin, two pieces of work which may appear to be in conflict with it can be considered.

Ranvier's paper[7] on the lymphatics in the rat's ear is said to show that the lymphatics in the dermis begin as blind diverticula. A careful reading of his paper discloses that it is a brief note, and that it contains one relevant illustration, which gives no microscopical detail and does not indicate the extent of the papillæ. Nevertheless this picture does present more continuous lymphatic loops than blind diverticula. There can be no doubt that a fuller study of this problem by Ranvier would have got rid of even this much discrepancy.

An account of the lymphatics of the skin has been written by Sampson Handley,[8] who used soluble Prussian blue for injection of the lymphatics in normal breast skin. The figure he gives (8) does not suggest a general occurrence of blindly ending diverticula of lymphatics in the papillæ of the human skin, as stated in the text, but it is consistent with the account of the skin lymphatics given in this paper. The study of thick sections is the only reliable method by which the continuity of loops and the occurrence of blind ends can be determined. This continuity is obvious in the illustrations and descriptions of such investigators as Teichmann,[9] one of whose figures is also reproduced by Piersol,[10] J. Neumann, v. Brunn, Unna,[11] and Bartels.[12] Yamao[13] really settled the point by his paper of 1932, in which he described his findings after lymphatic injection of human skin, both adult and fœtal and at different ages, and taken from most of the different regions of the surface. He showed what areas of adult skin and of skin from growing individuals actually contain blind lymphatic diverticula in the papillæ. It will be noted that his findings do not disagree with the account given here.

My investigations do not support Handley's idea[14] of discrete skin areas of small size each with an independent group of lymphatic vessels.

FUNCTIONAL BASIS FOR LYMPHATIC SPREAD OF CANCER

The Permeability and Strength of Lymphatics.—The functional basis for the lymphatic spread of cancer is as important as the structural, although many of its problems are abstruse and little advance has been made in connexion with them. The abundant evidence summarized by Drinker and Field[15] of the greater permeability of lymphatic than of blood-capillaries indicates that the former are readily permeable by colloidal particles such as the protein of blood-plasma, and by microscopic particles such as those of graphite suspensions or indian ink. Florey[16] has shown that these vessels also allow the rapid entry of red blood-cells into their lumina if inflammation is present, while Emminghaus[17] long ago showed that in venous obstruction red blood-cells pass in large numbers into peripheral lymphatics. These latter findings must be concerned with the transition from the phenomenon of permeability to that of rupture of the lymphatic capillary wall. Whatever physical process underlies such an entrance of red blood-cells into lymphatic capillaries, it is clear that Emminghaus's and Florey's instances bring home to us the facility with which it may be possible for cancer cells, lying extravasated as it were in the tissues, to find their way into the lymphatic capillaries if these are present. The relative rarity of lymphatic cancer emboli in microscopical sections may be due to the rapidity of their removal, and not to the infrequent occurrence of lymphatic embolism.

There is evidence that in regions where plexuses of lymphatic capillaries are rich these vessels are readily torn by injecting fluid into their immediate environment, that is, by mechanical forces. The immediate filling of large areas of the lymphatic plexus so often seen when investigators blindly inject lymphatics with various substances can only be explained by rupture of these vessels at many points (*see* Drinker and Field,[15] who also adopt this view). In such cases there is no accompanying filling of the blood-vessels. It seems, therefore, that lymphatic capillaries can be opened or ruptured readily by forces applied to them from outside. But strangely enough all authorities agree in giving these vessels great powers of dilatation to any internal distending force. One can speculate that perhaps the lymphatics do possess great elasticity in a direction transverse to their long axis, but very little in the direction of this axis. Such a hypothesis would in part explain the apparently conflicting facts, and would give meaning to the sinuosity of lymphatics where they cross the body joints (*see* Poirier[2]).

Next it can be stated as well established that massage greatly increases the chances of particles and cells entering lymphatic capillaries, whether they do so by altered permeability or by rupture of the vessels. Massage does not force injected particles into the blood-vessels. From the above remarks it will be seen that malignant lymphatic embolism will more readily occur than blood-vascular embolism providing lymphatic capillaries are present in the tissue concerned.

The rarity of lymphatic spread of ordinary sarcomatous neoplasms might be readily explained by the normal absence of lymphatic capillaries from the tissues which may undergo sarcomatous change.

The Rate of Flow of Lymph.—Once cancer cells have entered the lymphatic lumen the next controlling physiological factor must be the flow within these vessels. Unfortunately our knowledge of the actual rate of flow, continuity of flow,

and pressure of lymph in lymphatics is limited, and some important points are still disputed by those studying them. Thus, although most investigators agree that in spite of variations in magnitude there is some flow fairly continuously in all lymphatics (*see* the various tables and remarks in Drinker and Field's book[15]), Rouvière and Valette[18] have recently published a book in which they maintain that most lymphatic flow is discontinuous, periods of complete cessation of flow being followed by periods of rapid flow in response to exercise or massage. Rouvière has omitted to notice the direct observations of independent, practically rhythmical, contractions of valved lymphatics seen by Pullinger and Florey[19]; and in placing all the emphasis on extraneous adventitious factors such as fascial pulls, mechanical deformations by exercise of a part, intestinal movements, respiration, etc., as motor causes of lymph propulsion, he has been obliged to imply that the smooth muscle in the lymphatic wall is functionless. Rouvière appears to have accepted Funaoka's experiments[20] rather uncritically, and one feels on reading his book that this also has contributed to his unorthodox conclusions and to the unsatisfactory paragraphs on lymphatic contractility on page 73 of his book.

Perhaps the most reliable accounts of these subjects are to be found in the papers of Florey,[19] though one feels chary of accepting his negative finding of non-contractility of the true capillary lymphatics, when one takes into account the well-attested contractility of blood-capillaries. In the matter of definitely ascertained facts necessary for the problem of this paper, it has been observed that lymph can

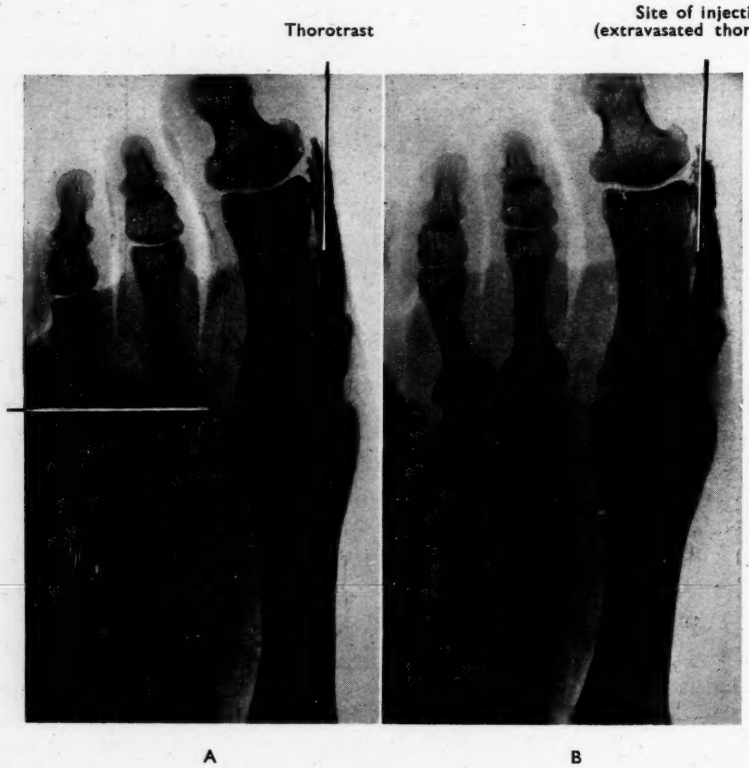


FIG. 319.—Radiographs of normal adult foot (No. 43). Thorotrast injections in the living. A, Taken immediately after injection and massage—subcutaneous plexus of great toe filled; B, Taken ten minutes later—lymphatic plexus now practically empty. X-ray exposure identical in the two pictures.

travel to the receptaculum chyli from the lymphatic capillaries in a dog's foot in ten seconds,[15] and observations made on the human subject (*Fig.* 319) show that the fine cutaneous plexuses of the skin of a toe and their draining trunks for the distance of the length of the foot can almost empty themselves completely in ten minutes or less time, when the foot and leg are left untouched, resting horizontally on a table. The great weight of evidence, both direct from such observations as those quoted, and indirect from our knowledge of the function of lymphatics in the body—to restore to the blood the important constituents lost from it at the blood-capillary stage of circulation[15]—leans to the view that the lymphatic vascular system is normally an active one exhibiting rapid and fairly continuous flow of its contained lymph. The rate of flow within the system is slower by mechanical necessity at the glands, where the rapid stream from one or more lymphatic trunks opens out into a 'lake' of sinuses. Incidentally, Rouvière's hypothesis[18] that the glands function as regulators of rate of flow, by swelling up if this be too rapid and gradually subsiding to normal size when the stress is removed, cannot be regarded as established.

One other fact is relevant to the present problem: it is that innumerable experiments and observations have convinced all investigators that lymph-flow is increased greatly by massage and exercise and general physiological activity of a part (Starling[21]).

The Fixed Direction of the Lymph Stream.—The almost universal presence of valves in lymphatics above the capillary stage fixes the direction of lymph-flow in these vessels, and since valve cusps can only carry out their function of preventing 'backflow' at instants when the forward flow has ceased, it might be thought that their presence implies periods of cessation of flow. But in this connexion it is necessary to bear in mind that a cessation need only be momentary to allow the valves to exert their function, and the transient halt need only involve a very short length of the vessel because of the relatively very great number of valves. The fixed direction of the lymph-stream and the assembling of lymphatic trunks from considerable areas of tissue at certain focal points are, broadly speaking, stable and inherited characters; and they enable us to foretell that lymph or other contents of lymphatics from a particular region or tissue of the body will be carried to particular lymph-glands. But actually there are in most regions certain more variable lymphatic pathways which either at the capillary or the lymphatic-trunk stage form anastomoses, which can lead some of the lymph into a different set of regional lymph-glands. From this point of view one can speak of the main or focal lymphatics and glands draining each tissue or region, in contrast to a collateral or subsidiary drainage for the same region. The subsidiary lymphatic pathways have been carefully investigated only for a few organs,[22] but there is evidence that they are more variable than the main ones, and that if the ordinary focal glands are blocked the subsidiary channels may multiply and become larger within limits (Rouvière[18]).

The effects of the valves and focal drainage may be counteracted to some extent by another factor—that is, the occurrence of incompetence of the lymphatic valves, with consequent retrograde flow of lymph. It has been the orthodox view that retrograde flow in valved lymphatics is very rare, and that the vessels will dilate to extreme and even rupture rather than allow reversal of the direction of flow. Retrograde injections have only rarely been achieved,[23] but Rouvière seems to be of the opinion that blockage of lymph-glands may fairly readily produce this

effect, particularly in collateral vessels, and he thinks that retrograde lymph-flow fairly often plays an important part in the re-establishment of lymphatic circulation after severing large lymphatic trunks by incisions.[18] He follows certain other authors in attributing importance to retrograde flow as a means of spreading infection from the neck into the lung and of carrying gastric carcinoma to the ovary. But it is difficult to find much evidence for such beliefs.

The focal drainage of the lymph and the number, position, and functional possibilities of anastomoses must be correlated with the picture of clinical lymph œdema. A general discussion, together with important experimental evidence on this matter, is given by Drinker and Field,[15] but in order to mention the relation of the questions of lymph œdema to those of cancer spread the following paragraphs have been included.

Lymph or other contents of the superficial dermal capillary lymphatic plexus can, in theory at least, pass from one region of the skin to any other cutaneous region of the body. But the existence of a continuous non-valved superficial plexus over the whole surface of the body does not in any way conflict with the well-known limitation of lymph œdema to the area of skin described as the cutaneous drainage field for any particular group of glands. It is the presence of the valved lymphatic trunks, large and small, coming from this plexus and running in directions highly specific for any one region of the skin which is responsible for the localization of lymph œdema when there is a block at the glands or in the trunks near the glands. At least this is the hypothesis to explain the well-established anatomical and clinical facts in this matter.

If one examines a case of lymph œdema such as that caused by axillary obstruction, it is seen that in the midline of the body, back or front, in the thoracic region the limit of the œdema, though clearly defined, is not an absolutely sharp line. There is a strip about ½ to 1 in. wide at the margin of the œdematous area in which the œdema gradually fades away. The size of the capillary lymphatics must limit the amount of fluid they can carry away per unit time, and this intermediate area of skin between skin which is completely œdematous and that which is quite free from œdema represents the amount of compensatory work the adjacent capillary lymphatic plexus in connexion with the trunks of a different drainage area can do to relieve the congestion. If there were numerous anastomoses at all levels between the larger lymphatic trunks draining the different fields of the skin, then lymph œdema could only be produced by blocking the thoracic duct, the right lymph-duct, etc. Or if the continuous non-valved lymphatic plexus of the superficial part of the dermis consisted of vessels having the calibre of the larger lymphatic trunks, or the capacity to dilate to such a size, then also lymph œdema could only be produced in the manner just described. But the superficial dermal plexus consists of capillary vessels only, and there is a limit to their capacity for carrying off lymph from an area drained by one set of trunks to that drained by a different set.

In regard to the spread of cancer it may be noted that stagnation of lymph-flow may disturb the usual course of malignant lymphatic emboli, and thus favour the occurrence either of lymphatic permeation or of secondary cutaneous cancer nodules. On these grounds permeation and secondary nodules should be late phenomena in the spread of cancer. The two conditions—altered lymph-flow and the existence of a superficial continuous non-valved lymphatic plexus—may explain all the characteristics of secondary cutaneous nodules.

The Regenerative Capacity of Lymphatic Vessels.—The problems of lymph œdema lead one to the questions of regenerative capacity of lymphatic vessels, and some of these probably have an important bearing on the spread of cancer. The growth of new lymphatic vessels during adult life may be very considerable, especially in certain circumstances, and E. R. Clark and E. L. Clark[24] have proved that it occurs by a sprouting of the endothelium of the capillary lymphatics. Severing the vessel is one of the adequate stimuli for such sprouting, but that it is not the only adequate stimulus is shown by the formation of extensive plexus networks of lymphatic capillaries in enlarged dermal papillæ as illustrated in *Figs.* 315 and 332. Incidentally it is difficult to reconcile these figures with the hypothesis put forward by Handley[14] that the papillary lymphatics in cutaneous papillomata are distended, and with his theory that skin cancer results from papillary lymphatic stagnation. The work of Pullinger and Florey[6] is also opposed to the opinions of Handley.

In the work described below there is some evidence for considerable alterations in the pattern of cutaneous and other lymphatics on the distal side of a cancer which happens to be placed in the pathway of lymphatic drainage so as seriously to interfere with this drainage (*Figs.* 331–335). Also there is confirmation of the statements of many other investigators dealing with new formation of lymph-glands when the ordinary regional lymph-glands have been largely destroyed by cancer deposits. These matters will be dealt with in the course of this paper, but for a discussion of much of the experimental work which has been done in this field one may refer to Rouvière.[18] Their bearing on the problem of lymphatic spread of cancer is probably very considerable.

FINDINGS IN THE PATHOLOGICAL MATERIAL USED

A total of 24 specimens presenting new growths was used for the experiments. All of these were removed at operation, so that none of the cases could be said to be inoperable. Two of the 24 proved to be non-malignant growths (both in the breast), and 2 of the remaining cancer cases were not successfully injected, this being due in one case to failure to realize the appropriate site for injection (a case of cancer of the colon), and in the other to the tissue being too long dead when the injections were done. The remaining 20 were all satisfactorily injected.

The sources of the cancers examined were: breast, 10; cutaneous epitheliomata, 4; rectum, 4; thyroid, 1; recurrent fibrosarcoma of the abdominal wall, 1.

After the injections with thorotrast, radiographs of the specimens were taken. Further and lengthy examination was then carried out after clearing the whole mass of each specimen. Thick and thin sections were cut, serial sections through some inches of thickness were made in some instances, and in many cases serial sections of blocks taken at frequent intervals from the centre of the cancer mass up to invaded lymph-glands were done, and the notes were supplemented by the plotting of accurate scale maps on squared paper with the aid of crossed wires placed in the ocular of the microscope and a microscope stage clamp with verniers attached. It is proposed to give here only very brief abstracts from the notes. This follows the course adopted for the first part of the paper, where most of the control work has been presented as an account without giving the protocols underlying each statement.

Tissue No. 6 (*Figs.* 320–324).—Spheroidal-cell scirrhus of breast; three months' history; tumour in upper and outer quadrant, loosely attached to skin and nipple, but not to deep fascia; extensive axillary gland metastasis; early skin nodule (4 mm. diameter) found;

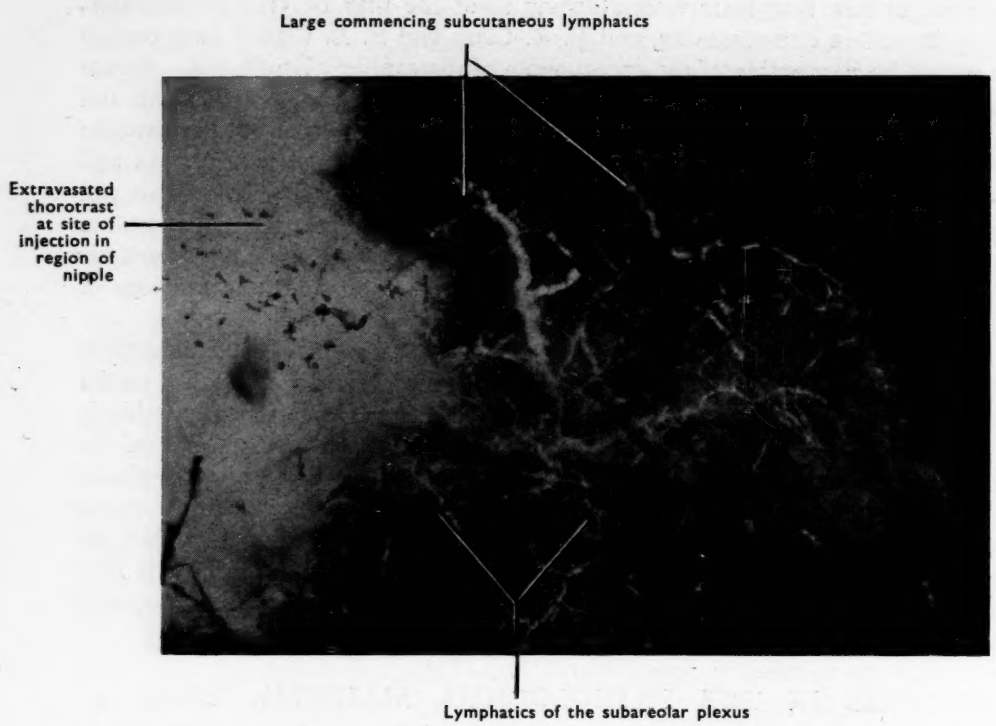

Large commencing subcutaneous lymphatics
Extravasated thorotrast at site of injection in region of nipple
Lymphatics of the subareolar plexus

FIG. 320.—Photograph taken with reflected light showing surface view of portion of skin overlying cancer of the breast (No. 6). Thorotrast injection of lymphatics. (× 4.)

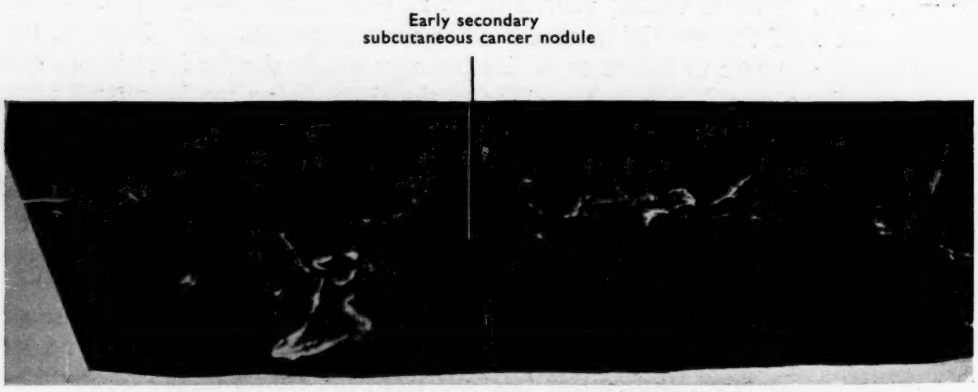


FIG. 321.—Photograph by reflected light of a thick section of same portion of skin as seen in *Fig.* 320. The cut surface of the section is seen. Note the secondary cutaneous nodule of cancer. (× 4.)

normal skin lymphatics superficial to the neoplasm, also normal lymphatics in subcutaneous tissue around neoplasm. At edges of the tumour there are normally patent lymphatics intimately surrounded by the neoplasm, but there are no lymphatics in the dense central parts of the tumour; no emboli or permeation found in lymphatic vessels.

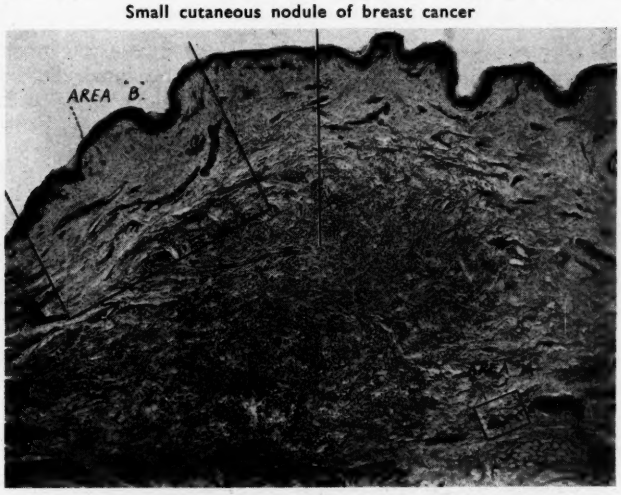


FIG. 322.—Section through secondary skin nodule shown in *Fig.* 321. (× 15.)

Scattered cancer cells infiltrating dermis and subcutaneous fat
Thorotrast-injected lymphatics typical of those surrounding the secondary skin nodule of cancer

FIG. 323.—Same section as *Fig.* 322, showing enlargement of area 'A'. (× 225.)

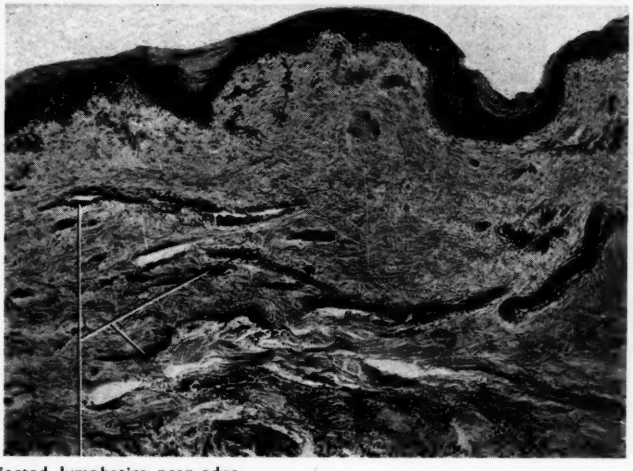


FIG. 324.—Same section as *Fig.* 322, showing enlargement of area 'B'. (× 42·5.)

Tissue No. 21 (*Figs.* 325–328).—Spheroidal-cell carcinoma of breast with extensive axillary-gland secondary deposits; tumour in upper and outer quadrant, fixed to skin not to fascia, 5 cm. diameter clinically; minute skin lymphatic plexuses filled in patches around

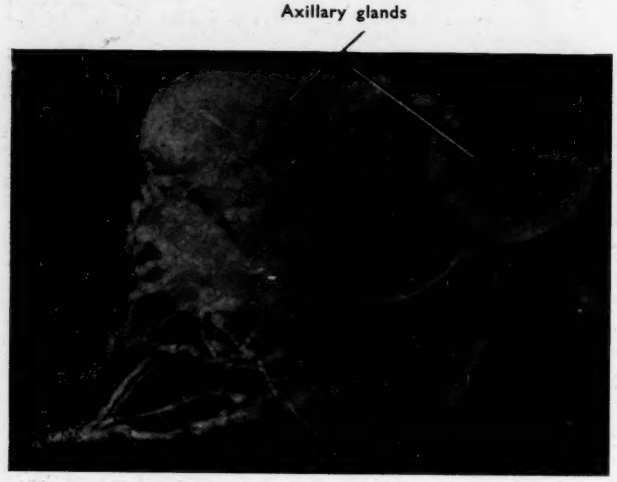


FIG. 325.—Photograph by reflected light of cleared specimen (No. 21) showing portion of axillary glands and lymphatic trunks. Large deposits present in these glands. (× 1½.)

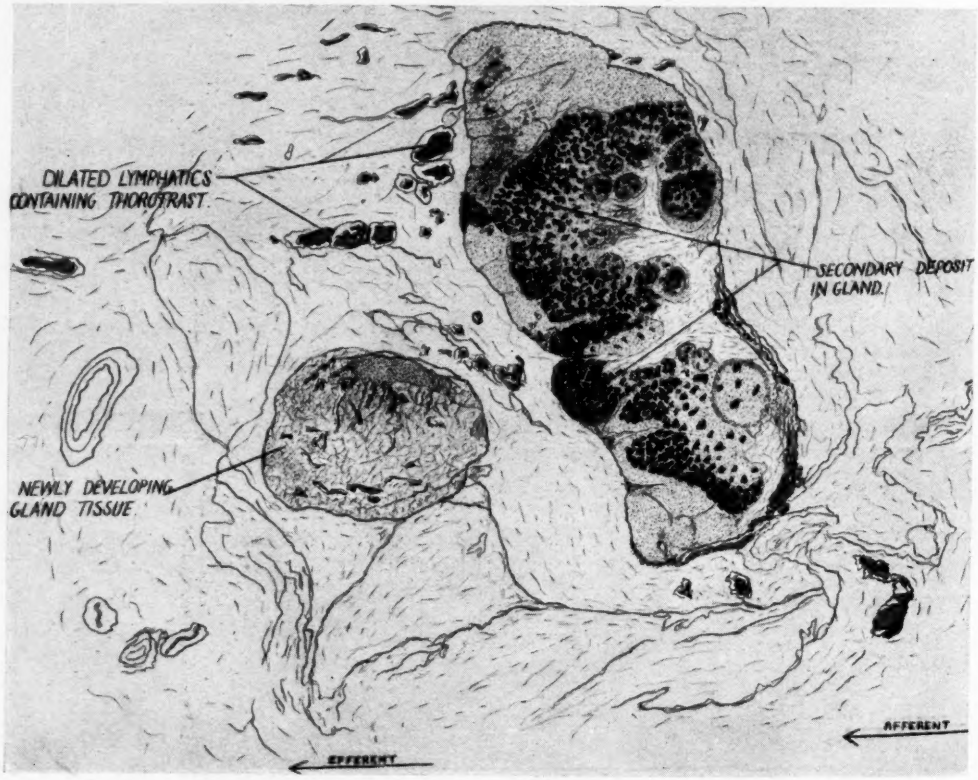


FIG. 326.—Microprojector drawing of section through axillary gland (No. 21). Large deposit of cancer in the gland. Dilated lymphatics. New gland tissue developing. (× 5.)

the areas of injection with thorotrast and are normal in appearance; the large afferent and efferent trunks related to axillary glands with extensive deposits appear normal in number,

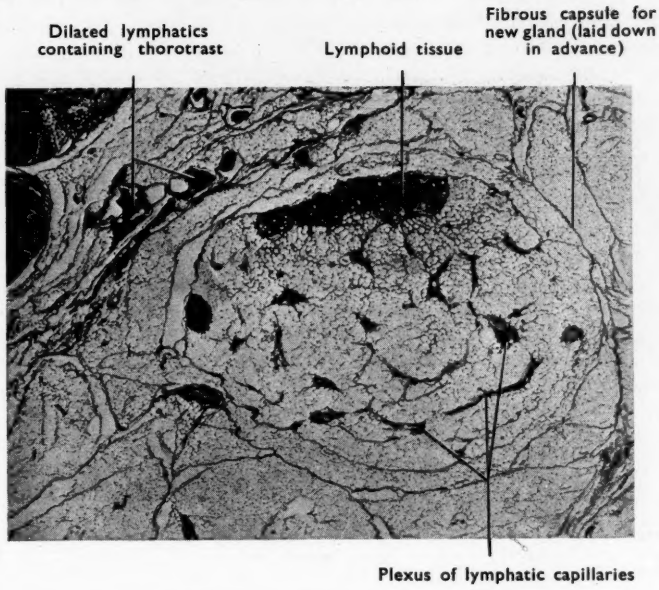

Dilated lymphatics containing thorotrast
Lymphoid tissue
Fibrous capsule for new gland (laid down in advance)
Plexus of lymphatic capillaries

FIG. 327.—Microphotograph of the newly developing gland tissue shown in *Fig.* 326. (× 10.)

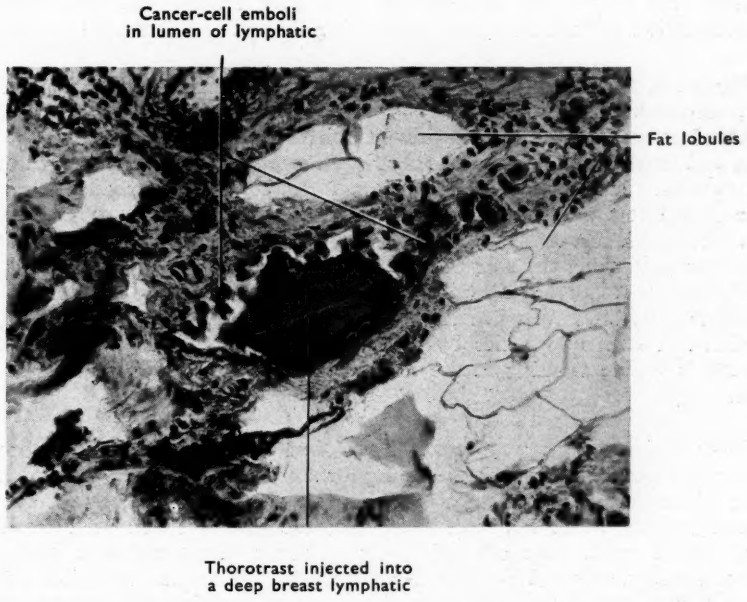


FIG. 328.—Microphotograph showing cancer-cell emboli in a deep breast lymphatic injected with thorotrast (No. 21). (× 190.)

patent in lumen but dilated; some lymphatic cancer-cell emboli, all small, consisting of only one or a few cells, found in efferent interglandular trunks from affected glands; permeation present for microscopic distances, but is confined within the limits of the infiltrative

spread of the growth; this permeation is patchy and far from complete; many lymphatics in the same positions are unaffected; small lymphatic cancer emboli found in some vessels near the growth; these consist of one or two cells; instances seen of small (naked-eye) lymphatic trunks completely unaffected by immediate contact of the cancer externally; skin lymphatic plexuses unaffected by extensive masses of neoplasm 1·25 cm. deep to them; instances of small (microscopic) lymphatic trunks embedded in cancer and suffering distortion and partial obliteration in patches by surrounding pressure; lymph-gland changes seem to point to new formation of gland tissue, both separate from and attached to the old gland. Is this the cause of lack of lymph œdema in many cases with extensive secondary deposits in glands?

Tissue No. 22.—Intraduct carcinoma of the breast; history of one year in a woman aged 27; tumour 2 × 5 × 1·5 cm., just below and external to nipple; fixed to skin but not to fascia; no deposits in axillary glands; small lymphatics of the breast gland itself have been well filled with thorotrast, as have the perivascular lymphatics; all lymphatics seem normal; no emboli found; no permeation found. The lymphatic capillaries all have their normal relationship to the glandular alveoli, being separated by a zone of connective tissue from immediate contact with the epithelium.

Tissue No. 24.—Scirrhous carcinoma of the breast from a woman aged 63; axillary-gland deposits present; breast had been amputated seven years earlier for Paget's disease; recurrence in upper third of scar for four months; clinically shows an indurated mass adherent to skin and muscle; patient died six months after operation, but there was no post mortem; the cancer has infiltrated a considerable amount of tissue, and a great deal of fibrous tissue has been laid down. The tissue was removed in several fragments so that the injections with thorotrast were partial only, but they show in a number of places normal lymphatics in close proximity to the scirrhus; some scattered instances of permeation are to be seen confined to areas of tissue which are extensively infiltrated by strands and masses of cancer cells among the fibro-areolar connective tissue; the total distances over which such permeation extends is microscopic, being limited to a few millimetres at the maximum.

Tissue No. 27.—Scirrhus of the left breast of a woman aged 48; small in size for three years; increase in size, fixation, and retraction of nipple for four months; no glands palpable, but secondary deposits present microscopically; tumour was just medial to the nipple, and fixed to skin and fascia; Halsted operation done, with no sign of recurrence after seven months. Examination of the injected cleared tissue showed a tumour about 10 c.c. in size placed at a depth varying from 0·5 to 2·0 cm. beneath the skin; several lymphatic trunks filled with thorotrast lie near this mass; one vessel coming from the skin overlying the tumour runs along the side of the tumour separated from it by less than 1·0 mm.; this vessel is quite normal; other vessels are seen running actually into the margins of the tumour, but soon disappearing in its interior, where there is no more thorotrast filling; sections show obliteration of such lymphatics by compression from the dense mass of cancer cells; one big trunk is visible running in the fat over the pectoral muscles towards the axilla; it is normal.

Tissue No. 31 (*Figs.* 329, 330).—Spheroidal-cell carcinoma of the breast in a woman aged 61; present for one year; fungating for six months; area of ulceration about 6 sq. cm. at time of operation; large tumour with peau d'orange of the breast skin and ulceration around the nipple, which has been destroyed; the growth is well fixed to the skin and fascia; axillary glands much enlarged and hard; opposite breast normal; operation was a local removal of breast, followed by deep X-ray therapy. Thorotrast injections filled the lymphatic plexuses and trunks at the periphery of the cancer and the subareolar plexus of lymphatics whose vessels were patent, though in some cases very much dilated; the cancer partly involves the subareolar region; plexuses of normal lymphatics are seen at the histological edge, and in some places actually within the carcinoma, which is more of the encephaloid type than scirrhous; there are a few instances of lymphatic permeation for minute distances at the advancing edge of the cancer mass in the subareolar region, otherwise no permeation found.

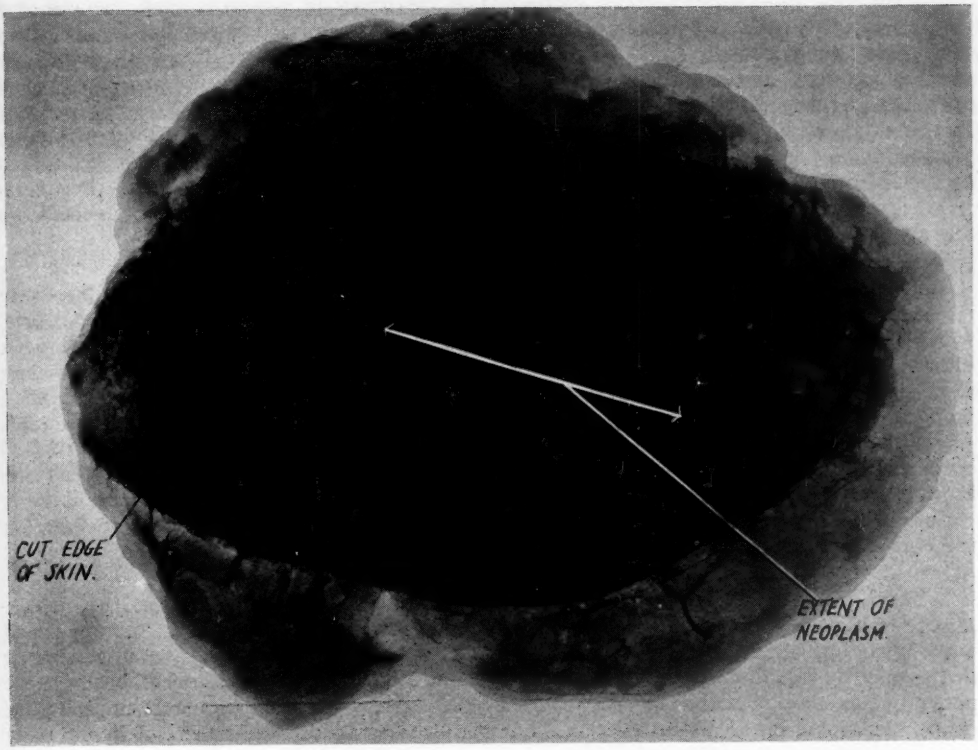


FIG. 329.—Radiograph of breast (No. 31). Large fungating carcinoma. Lymphatics injected with thorotrast. (× ⅔.)

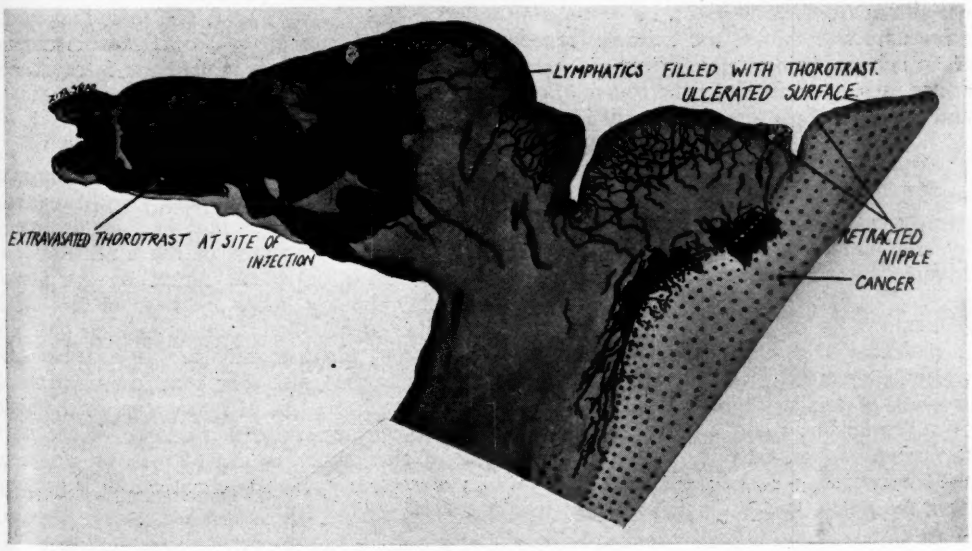


FIG. 330.—Drawing of hand-cut section from breast (No. 31). Lymphatic plexus adjacent to cancer. (× 7.)

Tissue No. 34.—Spheroidal-cell carcinoma of the breast in a woman aged 53, with five months' history; palpated tumour was about 6 cm. diameter placed in the axillary tail, fixed to the skin, but not to the fascia; it is a rapidly-growing cellular type of growth, with little stroma, frequent mitoses, and large axillary-gland deposits; treatment was by Halsted operation and deep X-ray therapy, and there was no recurrence six months later. Thorotrast injections showed patent normal lymphatics in contact with the edge of the growth; no permeation was seen; no emboli found; a number of the lymphatic trunks have been filled by the thorotrast, and these are all normal in patency though some appear dilated. Dilatation is more definite peripherally than near the glands. Skin lymphatic plexuses injected are not abnormal.

Tissue No. 38.—Scirrhous carcinoma of the breast, removed by local amputation; (?) deposits in axillary glands; mass of cancer about 4 × 4 × 2 cm. underlying the nipple, which is near one edge of the cancer mass; portions of the subareolar plexus have been filled by thorotrast, and are normal, the parts of this plexus not reached by the thorotrast show no invasion by lymphatic permeation as far as can be ascertained; one of the main draining trunks from the breast has been filled by thorotrast, and is seen coursing towards the axilla in the fat. It begins in the subareolar plexus, though its exact origin is masked by extravasated thorotrast; it runs over the growth in part of its course, at its closest being about 5 mm. away from it; it is completely unaffected by the cancer; the cancer is fairly close to the skin in some regions, near the nipple being about 1·5 cm. below the surface, but the cutaneous lymphatics are unaffected.

Tissue No. 39.—Spheroidal-cell carcinoma of the breast in a woman aged 52, with a history of six weeks; on palpation there was a lump about 5 cm. diameter in the upper and outer quadrant, unfixed; deposits present in axillary glands; there was a small recurrence in the scar six months after operation, which was a Halsted; thorotrast injections were not perfectly successful, but they showed that the skin plexuses of lymphatics were normal; the cancer mass lying in the fat shows several lymphatic trunks lying within 2 cm. of it, and these are completely unaffected by it. No permeation or emboli found microscopically.

Tissue No. 73.—Spheroidal-cell carcinoma of the breast, of a very cellular type, and with considerable axillary-gland metastases. The mass is about 5 cm. diameter, placed a little to one side of the nipple, which is retracted, and the growth is about 2 to 3 cm. below the skin surface; the lymphatics are unaffected by their proximity to the tumour, but as usual are absent from its interior; no permeation found. The axillary glands and their lymphatic trunks are well shown, and examples of new glandular formation are present, in two instances there being a chain of glands from pin-head size up to 2 cm. and more in diameter interpolated at irregular intervals along the course of a large lymphatic trunk. These masses are glandular, and none of those examined contained cancer deposits.

Tissue No. 9 (*Figs.* 331–339).—Epithelioma of leg near ankle, following very chronic varicose ulcer; spreading rapidly for eighteen months; deposit in popliteal gland for three months, now fungating; large deposits in inguinal glands for three months; leg removed by amputation through the middle third of the thigh. In the area of the ulcer there is compressive obliteration of lymphatics coming from areas below the cancer by the dense packing of epithelioma cells; there is dilatation of lymphatics below the level of this local obliteration by compression; extensive new formation of lymphatic capillaries is to be seen in the enlarged papillæ at the margins of the epithelioma; many of these papillæ have become warty excresences of considerable size (3 to 4 mm. in height), and they contain an extensive network of capillary lymphatics in their interior; there is no loss of lymphatics in the skin or subcutaneous tissue on the proximal side of the cancer; there is evidence of the new formation of lymphatic trunks on the distal side of the cancer, as can be seen by studying the illustrations; no permeation is to be found; a secondary skin nodule of cancer is present near the ankle, and has normal patent lymphatics all around it.

Tissue No. 25.—Epithelioma supervening on an old varicose ulcer of the lower leg (20 years), spreading rapidly for four months; biopsy of ulcer showed much hyperplasia

of epithelium, which in one area of the section showed definite change into a well-differentiated squamous-celled epithelioma; inguinal glands not examined microscopically, and remained as shotty eight months after operation as they were before; positive Wassermann reaction; the ulcer was 10 cm. wide, extending on the medial side of the leg from the sole of the foot upwards for 25 cm.; the operation was amputation through the middle of the thigh; there was no recurrence eight months after operation. Thorotrast injections showed a normal appearance of the lymphatics above the ulcer in spite of the considerable thickening

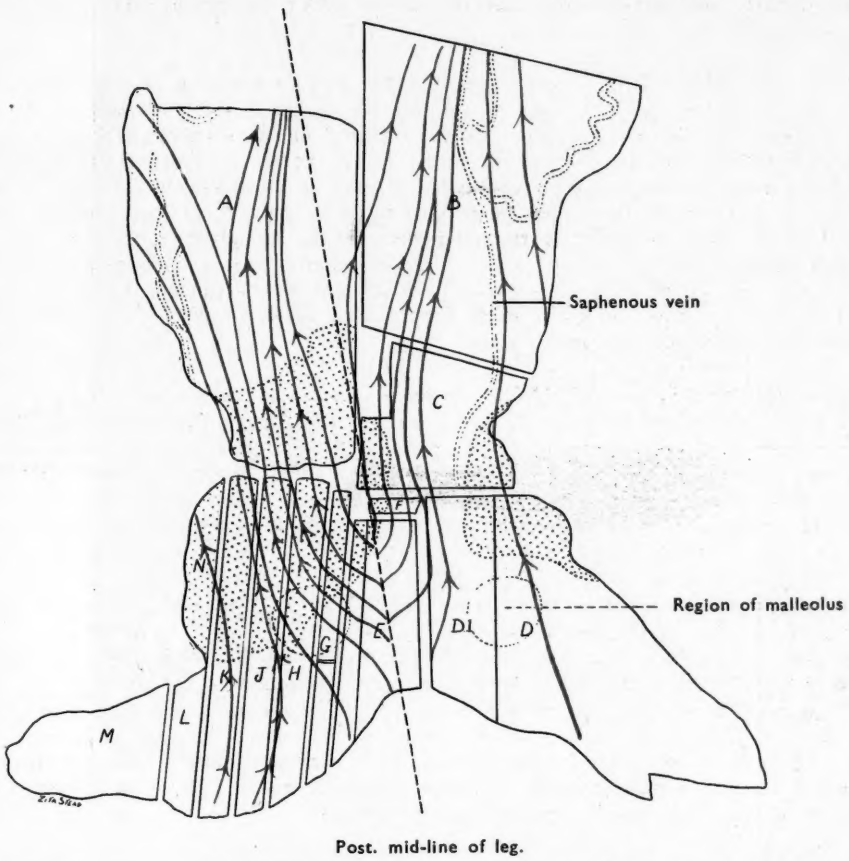
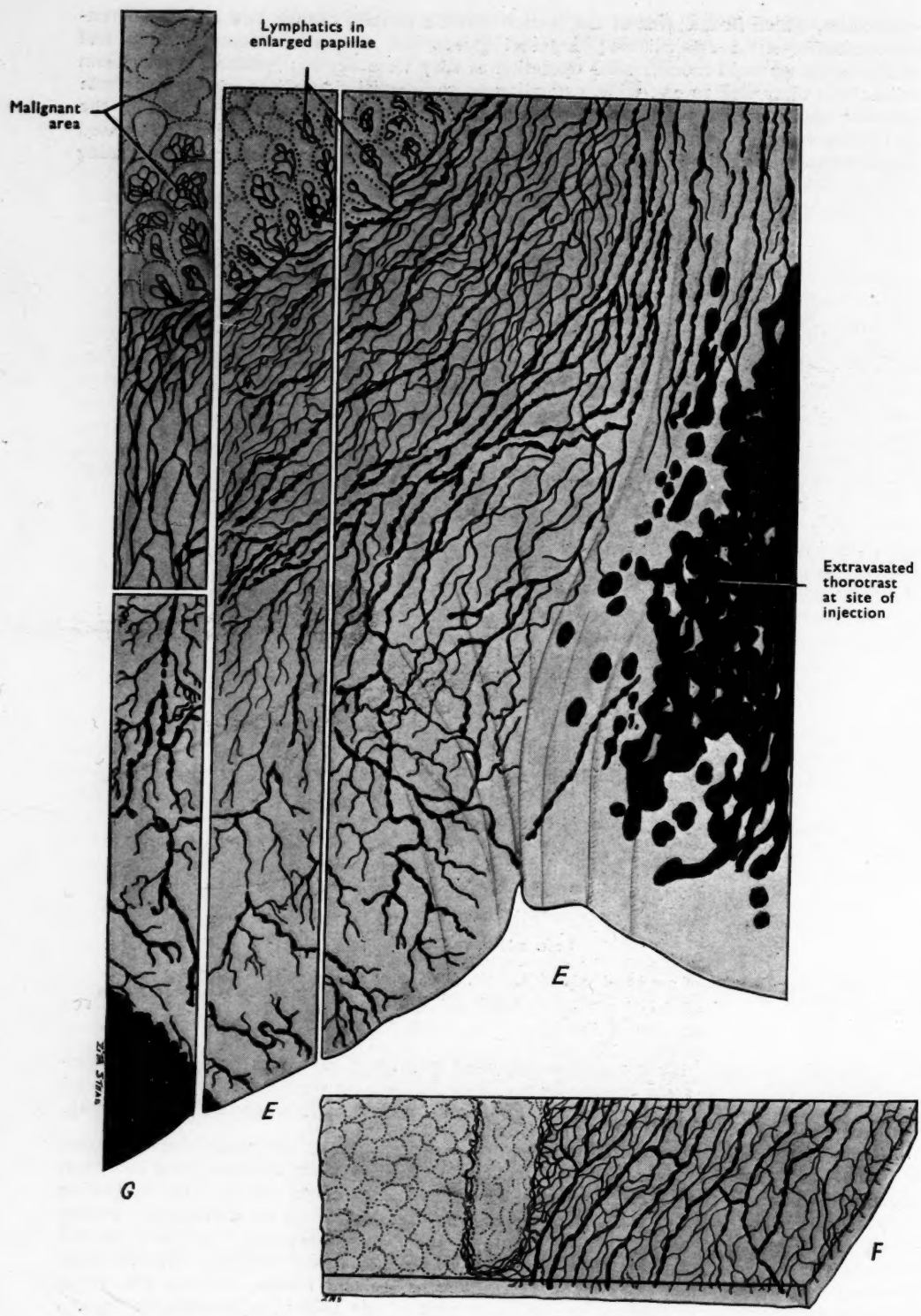


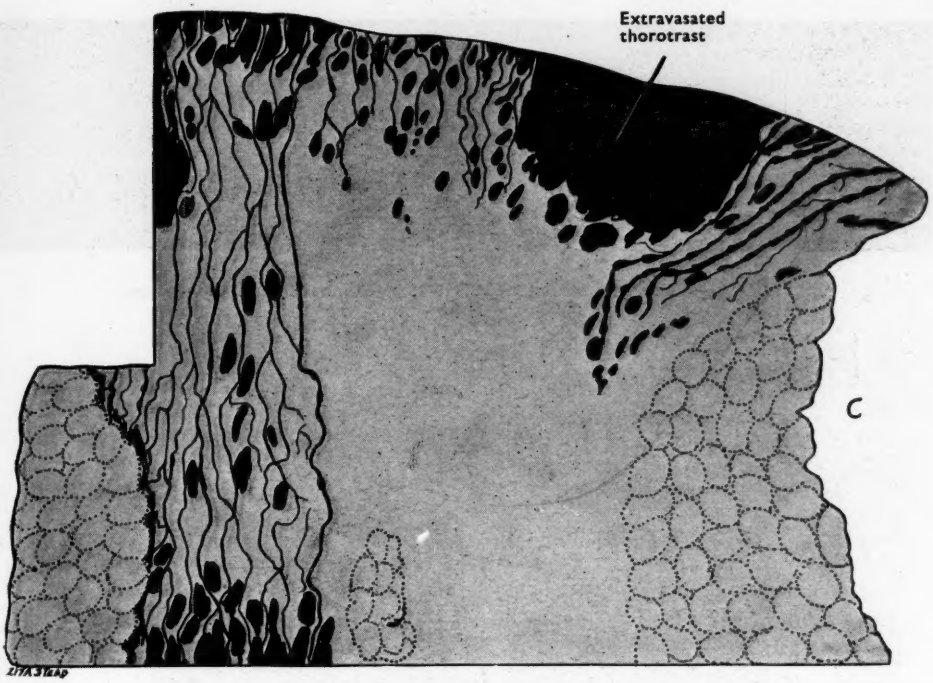
FIG. 331.—Diagram of outspread skin of leg (No. 9) to show area of ulcer (dotted) and manner of subdivision of skin. Red lines show the course which normal subcutaneous lymphatic trunks would take.

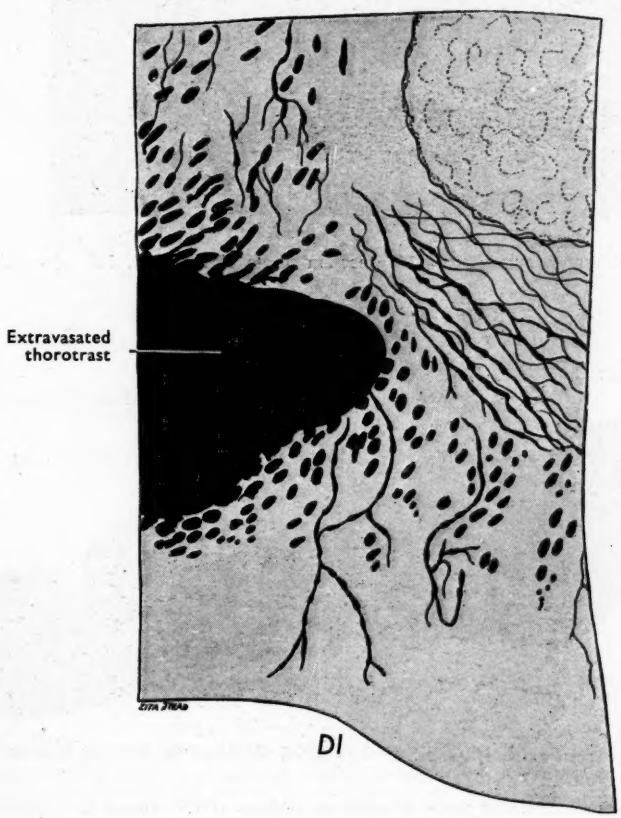
of the dermis present; the lymphatics appeared mostly of fine calibre; papillary overgrowths were present in some regions as in *Tissue No.* 9; no permeation or emboli found anywhere; there was the usual absence of lymphatics from the dense interior of the growth.

Tissue No. 47.—Squamous-cell carcinoma of the ear, recurrent two years after treatment by deep X-ray therapy; ulcer had then about two years' previous duration, and had been diagnosed by biopsy; the growth is a well-differentiated epithelioma, chiefly of the burrowing type, with little production of warty excrescences, but the surface has been ulcerated to some extent, chiefly in the lower and posterior part of the helix. Thorotrast injections showed lymphatics in the usual relation to the cancer-cell masses, a very complete picture being presented by both the thick and thin sections; vessels are seen leading off from the region of the growth particularly running in the upper part of the helix; no permeation found; clinically no gland deposits.



FIGS. 332, 333.—Freehand drawings of lymphatics seen in portions of skin shown in *Fig.* 331.





FIGS. 334, 335.—Freehand drawings of lymphatics seen in portions of skin shown in *Fig.* 331.

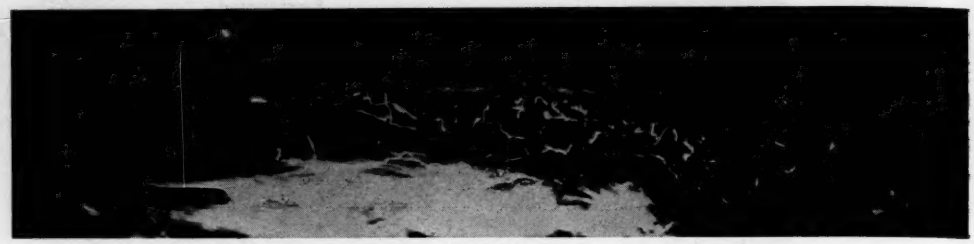


FIG. 336.—Photograph by reflected light of a thick hand-cut section across piece *F*, *Fig.* 333. (× 10.)

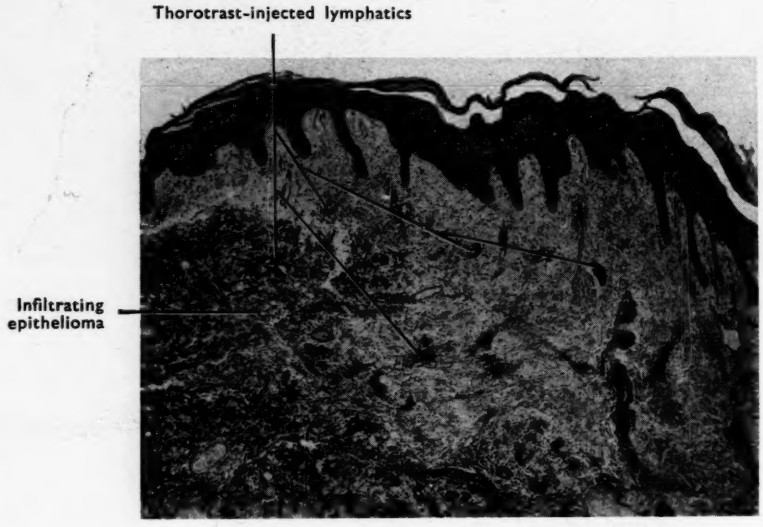


FIG. 337.—Microphotograph of a 12 μ section across piece *F*, *Fig.* 333. (× 42.)

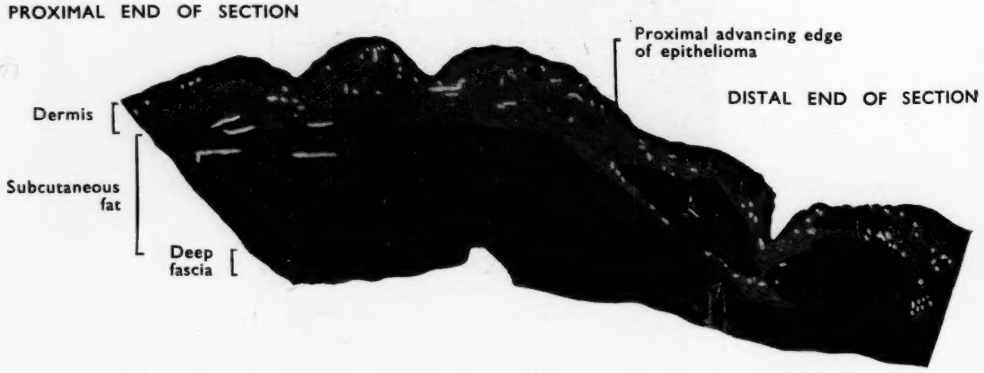


FIG. 338.—Map of distribution of lymphatics in relation to advancing proximal edge of epithelioma (No. 9). Lymphatics shown in white, only those of the dermis being plotted. All points were plotted accurately, using verniers. The lymphatics shown were all normal and injected with thorotrast. No permeated vessels present. The field covers about 2 cm. of fixed skin. Section 15 μ thick.

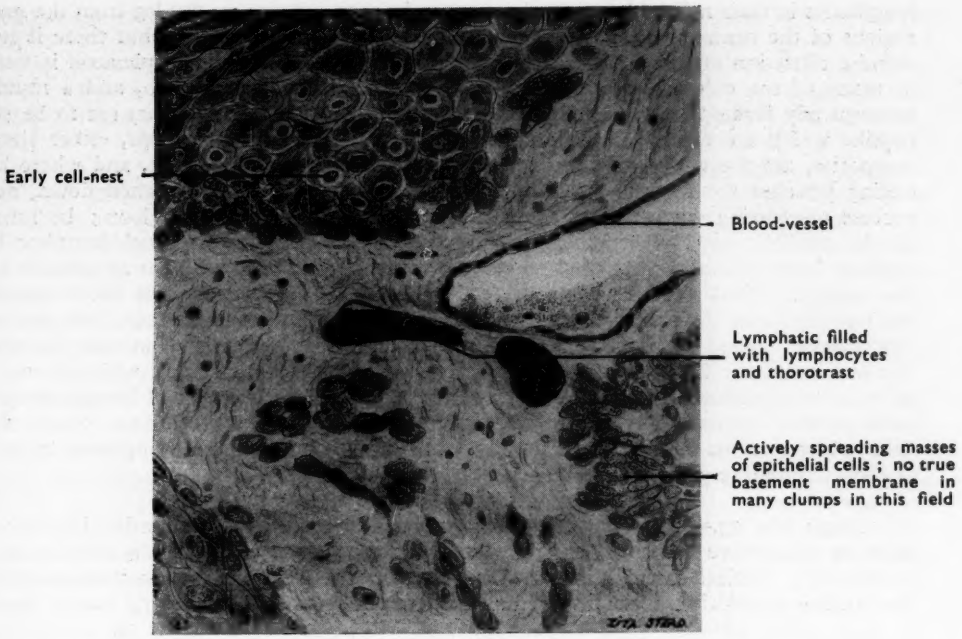

Early cell-nest
Blood-vessel
Lymphatic filled with lymphocytes and thorotrast
Actively spreading masses of epithelial cells ; no true basement membrane in many clumps in this field

FIG. 339.—Drawing from a section at the lower margin of an epithelium of the leg (No. 9) showing relation of lymphatics to epithelial masses. Camera lucida. (× 250.)

Tissue No. 50 (*Fig.* 340).—Epithelioma of the foot and ankle, following on a varicose ulcer which had been present for fifteen years in a woman aged 49. Ulcer was about 7 cm. diameter, and placed on the inner side of the ankle and heel, nearly extending on to the side of the foot. Radium needles and radon seeds had been inserted nine months before the

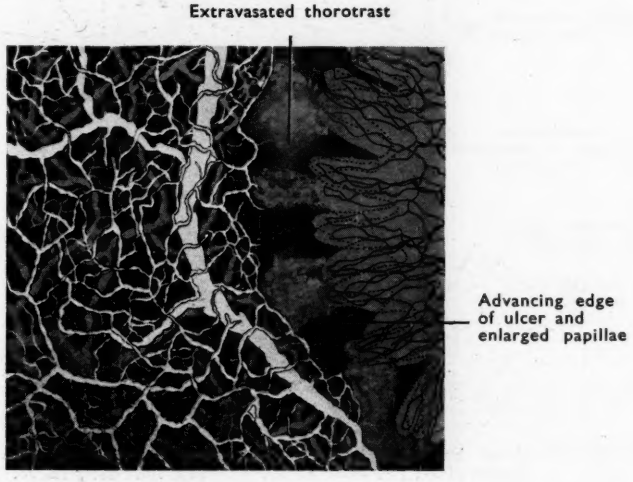


FIG. 340.—Drawing of edge of epithelioma of leg (No. 50). Shows adjacent lymphatic plexus thorotrast injected). Some of the injected blood-vessels (indian ink) are also shown. (× 10.)

leg was amputated, at which time there were several glands in the groin involved ; sections showed a squamous-cell epithelioma of Grade I type with well-keratinized cell-nests ; injections of the blood-vessels were done with indian ink and the lymphatics with thorotrast ; the

lymphatics in their normal large numbers are to be seen running up the leg from the marginal regions of the cancer; these trunks appear a little larger than normal, but there is no very definite dilatation of them. The new formation of capillary lymphatic plexuses is well seen in many of the enlarged dermal papillæ at the margins of the growth, and a number of incompletely formed loops (that is, blindly ending diverticula or pouches) are to be seen in papillæ which are chiefly of the long, narrow type; but complete loops, either simple or composite, are the commonest finding in these enlarged dermal papillæ, and where blindly ending pouches are present the free end of the vessel is generally turned down, as if in process of growing down to join the plexus and thus form a complete loop; by no means all the papillæ contain lymphatics, but all the papillæ contain the usual complete blood-capillary loop, which in most of these cases is coiled like a spring where it approaches nearest the surface. Thick sections show perfectly the distinction between the blood-vessels and the lymphatics of the skin. There are instances of permeation to be seen, but nowhere do they extend beyond the infiltrated area, and they seem very occasional in their distribution, just one lymphatic here and there being permeated for a short distance, while all round and about such a permeated vessel are to be seen the normal non-permeated lymphatics in their usual normal concentration; lymphatics well within the growth are absent, but in the less dense outer margins they are to be found; compressive obliteration of lymphatics by pressure of the surrounding growth is to be seen.

Tissue No. 23.—Columnar-cell carcinoma of the rectum; six months' history; large ulcer 12 cm. above the anus, encircling the gut completely except for a strip 2 cm. wide posteriorly; vertical extent of ulcer about 6 cm.; the removed rectum was opened along the midline anteriorly; ulcer involved all coats of the rectum in the central parts; thorotrast

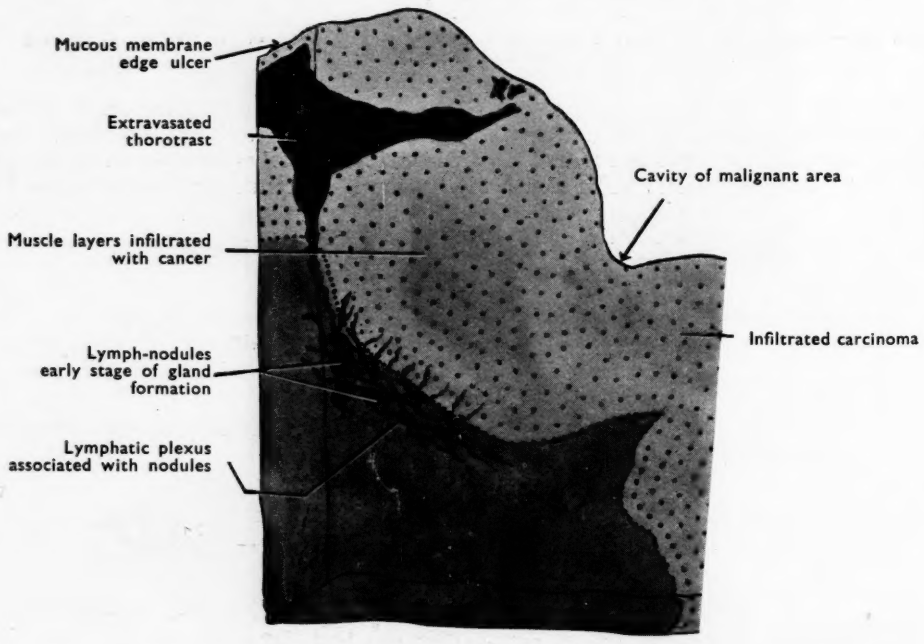

Mucous membrane edge ulcer
Extravasated thorotrast
Cavity of malignant area
Muscle layers infiltrated with cancer
Infiltrated carcinoma
Lymph-nodules early stage of gland formation
Lymphatic plexus associated with nodules

FIG. 341.—Freehand drawing of hand-cut section at edge of rectal cancer (No. 23). Shows new lymph-gland formation in the perirectal fibro-fatty tissue. (× 7.)

injections were made around the edges of the ulcer within and just outside its margins; a good part of the rich plexus of perirectal lymphatic trunks and glands was thus filled; lymphatic plexuses were present and normal right up to the margins of the cancer; there are secondary deposits in glands of the perirectal region close to the ulcer, but although there are over thirty gland masses present very few of these contain deposits; the vast majority

of the glands are small, round, probably newly formed, and contain large lymphatic capillaries with ordinary endothelium, appearing very different from the typical normal lymph-sinuses; what may be early stages of this new gland formation are represented in *Fig.* 341; no permeation found in this specimen. A radiograph of this specimen is published in Sir C. Gordon-Watson's article in *The Lancet*.[25]

Tissue No. 35.—Adenocarcinoma of the rectum in a man aged 39, with ten months' history; the growth was ulcerated and involved the whole circumference of the rectum, being placed about 10 cm. from the anal margin; liver normal clinically; patient died four days after the operation. The growth was a poorly differentiated one, with deposits in the pararectal glands; after thorotrast injection numerous ascending lymphatic trunks coming from the region of the ulcer were seen, and the usual findings of close relation of normal lymphatics to the margins of the growth were obtained. Two cases of proved lymphatic embolic involvement of glands by secondaries were found; one was a gland 2 cm. above the proximal margin of the cancer, which contained a small deposit visible through 51 sections each 10 μ thick; the serial sections showed that all lymphatics, afferent and efferent, connected with this gland were normal; a second gland about the same distance from the primary cancer showed a very early deposit (visible in four consecutive sections—an embolus of about 15 cells caught in the peripheral sinus of the gland); the first embolus was also growing in the periphery of the gland; many infiltrating columns of cancer cells were seen in the mucosa at the margins of the ulcer; these could all be traced back to the main mass of the cancer, and where these outliers were enclosed in a space it was shown to be a shrinkage space and not a lymphatic; no permeation found.

Tissue No. 45 (*Fig.* 342).—Adenocarcinomatous ulcer of the rectum in a man aged 59, with one month's history; the ulcer was low down in the rectum on its front wall, about 8 cm long, and not fixed; X-ray examination showed a deposit in the 4th lumbar vertebra; there were deposits in the pararectal glands, one of which was almost completely destroyed, yet

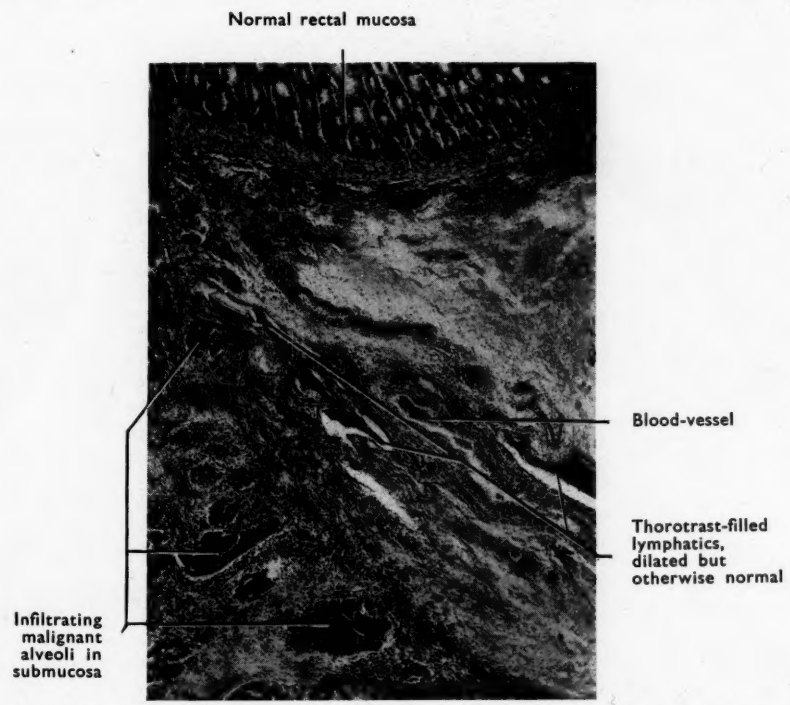


FIG. 342.—Microphotograph of section through rectum (No. 45) near edge of adenocarcinoma. Normal mucosa above, infiltrating malignant alveoli in lower corner of field. Dilated lymphatics (thorotrast filled) in submucosa in centre of field. (× 22.)

there was no permeation of vessels joining it; the thorotrast injections showed cancerous areas of the mucosa, with many dilated patent lymphatics between the irregular glandular acini; some of the lymphatics around the gland which had been practically destroyed showed early stages in thrombosis, but no permeation; patient died seven months after operation, but there was no post mortem.

Tissue No. 74.—Poorly differentiated adenocarcinoma of rectum, involving all coats of the rectum, and presenting a large ulcer about 8 cm. diameter, and with its lower margin about 5 cm. above the anus. Deposits present in pararectal glands; at its margins the cancer has not involved the muscular coats completely; thorotrast injections showed the usual picture of patent normal lymphatics in relation to the margins of the cancer, and their absence in the cancerous areas, where great irregular masses of cells were present; in the submucosa and in the perirectal tissue close to the cancer there is the appearance of proliferation of lymphatic capillaries and laying down of lymphoid masses; farther out in the perirectal tissue the rich plexus of lymphatics and glands presents the characteristic appearance of cases of rectal cancer; no permeation found.

Tissue No. 54.—Carcinoma of the thyroid gland in a woman aged 56, with history of a goitre for five years previously; the tumour was a hard mass about 5 cm. in diameter placed in the right lateral lobe, two smaller masses of similar consistency being present below the main one; there was no hyperthyroidism; X-ray diagnosis of secondary deposits in the ribs and (?) in the lungs; at operation the tumour and surrounding parts of the gland (lateral lobe) were removed; thorotrast injections showed well many parts of the lymphatic plexus in the gland and on its surface; large areas of the gland are replaced by cancer which shows

FIG. 343.—Photograph of hand-cut section of recurrent fibrosarcoma of abdominal wall. Thorotrast injection of vessels (? lymphatics) within the substance of the tumour (No. 36). (× 4.)

some differentiation into vesicles, but these are very incomplete and ill-formed; the lymphatics seem unaffected in the non-cancerous areas, even when quite close to the malignant patches (within a centimetre); in the interior of the big patches of cancer there are no lymphatics filled by thorotrast; but at the edges of these patches there are lymphatics to be seen becoming less numerous and more distorted as they extend farther into the interior of the large cancer masses. Microscopically one sees distortion and dilatation of lymphatic capillaries; in many places there is intimate contact of cancerous vesicles with lymphatic capillaries; sometimes this is associated with partial destruction of the lymphatic wall (gaps being present), and often with gross distortion of the vessels; no permeation seen; some small emboli seen in some of these lymphatic capillaries.

Tissue No. 36 (*Fig.* 343).—Recurrent fibrosarcoma of the abdominal wall in a man aged 49; the growth, adherent to the spermatic cord, but not to the skin, had been removed many times during fifteen years; at the last operation the growth was removed, and was received for thorotrast injection as an encapsulated mass about 6 cm. diameter. The injections showed a very extensive network of vessels with very thin walls and irregular lumen with pouch-like dilatations; they did not appear to be typical lymphatics; sections failed to reveal any valves in these vessels, but also there was no blood in them; in an area which had undergone hyaline degeneration these vessels did not fill with thorotrast, and so a sharp line of demarcation separated the degenerated area devoid of thorotrast in its vessels from the surrounding area of the growth which showed the peculiar vessels filled with thorotrast; these vessels may correspond to poorly developed lymphatics, and seem to resemble the spaces shown by Borst in a somewhat similar tumour.[27]

DEDUCTIONS FROM THE PATHOLOGICAL FINDINGS

The deductions from these investigations may now be summarized along the same lines as those used in the British Empire Cancer Campaign Reports for 1936 and 1937,[3, 27]:—

1. The mode of spread of cancer to lymph-glands is generally by means of lymphatic emboli of cancer cells carried along in the lymph-stream.

2. The results indicate that cancer of its own accord does not generally stimulate new formation of lymphatic vessels, except in cases where:

a. There is substantial formation of stroma belonging to the cancer or associated precancerous changes involving the laying down of new connective tissue in relation to epithelium (e.g., enlarged dermal papillæ).

b. Lymph-glands are interfered with, either by the presence within them of cancer deposits or by the abnormal state brought into being in some unknown manner by the presence of a cancer peripherally. The new formation of lymphatics in this case is central or focal—that is, occurs chiefly in the region of the focal lymph-glands. Also it is associated with the formation of new lymph-glands by the subsequent changes occurring around the newly formed plexuses of lymphatic capillaries. Further work is necessary to provide proof for the phenomena described under this heading, but the evidence obtained points to its being correct.

3. The above statements are not meant to imply that there is any evidence from this work for a lymphotropic theory of cancer. The ordinary effects of cancer in appreciable masses upon the local lymphatic vessels are not lymphotropic but purely indifferent mechanical effects, leading ultimately to compression and disappearance of the lymphatic vessels within the interior of a cancer. Lymphatic thrombosis plays very little part in these phenomena.

4. Cancer cells generally do not remain for any length of time within the lumen of a lymphatic vessel; that is, the spread of cancer cells by their growth as cords in the lumina of lymphatics is an unusual phenomenon in cases of operable cancer.

When it does occur such permeation seems to be generally only of microscopic extent. The claim that destruction of permeated lymphatics and their contained cancer cells by thrombotic changes does occur could not be confirmed.

5. On the grounds of anatomy and physiology the focal lymph-glands will be the first and most likely seat of glandular cancer metastasis. But subsidiary or collateral glands may also be involved, though it is to be expected that this will follow later than involvement of the ordinary focal lymph-glands.

THE LITERATURE ON LYMPHATIC SPREAD OF CANCER

There is an extensive literature on the lymphatic spread of cancer, and it has been carefully considered. It may be subdivided into work in which the lymphatics concerned have been injected, and work in which this and other identification tests for lymphatics were not done. Of the former work, the findings of Krause,[28] H. M. Evans,[29] F. C. Lee,[30] and of E. E. Goldmann,[31] agree completely with the results here described. But Langhans in 1875,[32] after injecting lymphatics in cases of breast cancer, came to the conclusion that the perivascular lymphatics may be extensively filled with cancer cells, and he gave a drawing illustrating this. The conclusion from the present work is that such an event is rare among operable cases of cancer. Of the great mass of work in which injections were not done and lymphatic identification tests were incomplete, some workers agree with the present findings and some do not.

Limitations of space prevent further discussion of the literature, though it may be mentioned here that the paper by Pullinger and Florey[6] gives a fairly full discussion of the literature on the relation of lymphatics to cancer, particularly of literature on experimental work.

GENERAL SUMMARY

A new method of injecting lymphatic vessels has been developed in which thorotrast is used, and this method allows of study of the lymphatic system under a very wide range of conditions.

The method has been applied to 20 cases of operable cancer backed by control experiments on normal tissues, and in some cases associated with injection also of the blood-vessels; and in the analysis care has been taken to use adequate methods for the problems associated with the lymphatic spread of cancer.

Briefly it may be said that this and other work adequate for the solution of the problem of lymphatic spread of cancer demonstrate that for operable cases of cancer embolism is the only important method, and that lymphotropic theories of cancer are incorrect, as are lymph-stases theories of the origin of cancer. The contributions which the basic medical sciences make to the pathological and clinical problems of lymphatic spread of cancer have been considered.

Acknowledgements.—It is impossible for me to acknowledge individually in print my gratitude to all the many persons who have helped me so much, and made this work possible. But it is true that whatever value the work will have is due chiefly to Professor H. H. Woollard, to whom is due the suggestion for the thorotrast method, and to whom I owe my introduction to many of the surgeons of St. Bartholomew's Hospital, who so kindly provided the cancer material, and to

Dr. G. Simon who did the bulk of the radiological work. Also to Professor Woollard I am indebted for continuous help and advice during the three years in all the methods of research involved. While at St. Bartholomew's Hospital, where the greater part of the work was done, my position was that of Cancer Research Fellow working under the Cancer Research Committee of the Hospital. To the members of that Committee I am much indebted for their kindly help and encouragement. During 1937 at University College, London, the expenses of the work were defrayed by a grant from the British Empire Cancer Campaign, to whom accordingly my thanks are due. To Miss Stead, the anatomical artist, I wish also to express my gratitude.

REFERENCES

[1] GRAY, J. H., *Jour. of Anat.*, 1937, lxxi, July.
[2] POIRIER, P., et al., *The Lymphatics*, English translation by C. H. LEAF, 1903. London: Constable.
[3] GRAY, J. H., *Annual Report of British Empire Cancer Campaign*, 1936, 34.
[4] SAPPEY, M. P., *Anatomie, Physiologie . . . des Vaisseaux lymphatiques . . .* 1874. Paris.
[5] ROUVIÈRE, H., *Anatomie des Lymphatiques de l'Homme*, 1932. Paris.
[6] PULLINGER, B., and FLOREY, H., *Jour. Pathol. and Bacteriol.*, 1937, xlv, 157.
[7] RANVIER, *Comptes rend. de l'Acad. des Sci.*, 1895, cxxi, 856.
[8] HANDLEY, W. S., *Cancer of the Breast and its Operative Treatment*, 1922. London: John Murray.
[9] TEICHMANN, *Das Saugadersystem*, 1861. Leipzig.
[10] PIERSOL, G. A., *Human Anatomy*, 1907, *Fig.* 783. Philadelphia: J. B. Lippincott Co.
[11] UNNA, P., *Arch. f. mikr. Anat.*, 1908, lxxii, 161.
[12] BARTELS, P., *Handbuch der Anatomie des Menschen* (v. Bardeleben), vol. iii, pt. iv.
[13] YAMAO, O., *Folia Anatomica Japonica*, 1932, x, Pt. 4, 505.
[14] HANDLEY, W. S., *The Genesis of Cancer*, 1931. London: Kegan Paul.
[15] DRINKER, C. K., and FIELD, M. E., *Lymphatics, Lymph, and Tissue Fluid*, 1933. London: Ballière, Tindall & Cox.
[16] FLOREY, H., *Jour. of Physiol.*, 1927, lxiii, 1.
[17] EMMINGHAUS, H., Ludwig's *Arb. a. d. physiol. Anstalt zu Leipzig*, 1873, viii, 51.
[18] ROUVIÈRE, H., and VALETTE, G., *Physiologie du Système lymphatique*, 1937. Paris: Masson et Cie.
[19] PULLINGER, B. D., and FLOREY, H. W., *Brit. Jour. Exper. Pathol.*, 1935, xvi, 49.
[20] FUNAOKA, S., et al., *Arb. a. d. anat. Inst. d. kaiserlich. Univ. Kyoto*, 1930, Ser. D., pt. 1.
[21] STARLING, E. H., *Jour. of Physiol.*, xvi, 224.
[22] LEVEUF, J., and GODARD, H., *Rev. de Chir.*, 1923, lxi, 219.
[23] LEE, F. C., *Contrib. to Embryology* No. 74, Carnegie Inst. Vol. xv, 1923.
[24] CLARK, E. R., and CLARK, E. L., *Amer. Jour. Anat.*, 1932, xlix, 441.
[25] GORDON-WATSON, C., *Lancet*, 1938, i, 239.
[26] BORST, *Die Lehre v. d. Geschwulsten*, 1902, vol. 1, pt. xv, *Fig.* 83. Wiesbaden.
[27] GRAY, J. H., *Annual Report of British Empire Cancer Campaign*, 1937, 178.
[28] KRAUSE, W., *Deut. Klin.*, 1863, xv, 377.
[29] EVANS, H. M., *Bull. Johns Hopkins Hosp.*, 1908, xix, 232.
[30] LEE, F. C., TILGHMAN, R. C., and CARMICHAEL, R., *Arch. of Surg.*, 1933, xxvi, 1, 602.
[31] GOLDMANN, E. E., *Beitr. z. klin. Chir.*, 1911, lxxii, 1.
[32] LANGHANS, TH., *Arch. f. Gynäkol.*, 1875, viii, 181.

INTRAPELVIC CYSTS, PROBABLY OF MESONEPHRIC ORIGIN

By A. R. H. OAKLEY, London

In the following article a case is reported of two pelvic cysts in the male, probably of mesonephric origin. The recorded cases have been collected, an analysis made of the more recent ones, and the subject of mesonephric cysts as a whole is discussed.

CASE REPORT

History.—The patient, an unmarried male aged 18 years, was admitted to hospital on a Sunday morning, sixty hours after the commencement of his symptoms. On the Thursday previous to admission, between 6 and 10 p.m., he had diarrhœa, defæcating three times, the act being associated with central abdominal pain of a mild nature. He slept well. On the Friday he defæcated three times, with central abdominal pain of a shooting and intermittent type, which settled in the right iliac region. He slept well again, and the next day had no pain, and passed no motion. During the Saturday night the pain recurred, was more severe, and was confined to the right iliac region. On the Sunday, the day of admission, he vomited at 7 a.m., and this was the only occasion. He defæcated once, and had slight frequency of micturition. He had been in bed since the onset of his symptoms. His stools were not offensive, nor otherwise abnormal.

During the last year he had had six similar attacks of pain, but never went to bed. No operations. Lobar pneumonia in 1934.

No brothers or sisters. Mother and father well; no operations. As far as he knew, he had no relatives who had had any unusual illness or operation.

On Examination.—The patient was a normal healthy male. Temperature 101° F. Pulse-rate 100. The tongue was furred. The routine urine tests were within the average standards.

The right iliac abdominal region was tender, and muscle guarding was present. There were no left-sided signs, and no pathological intra-abdominal mass was palpated. The external genitalia were developed fully and the intrathoracic organs were in their customary position. Per rectum a spherical mass the size of a golf-ball could be palpated through the left rectal wall, an index-finger's length up. It was slightly tender, compressible, and fixed to the pararectal tissues, but not to the rectum itself. The prostate was of average size and consistency, and no connexion was apparent clinically between this and the mass.

A diagnosis of a subacute pelvic peritonitis, originating in the appendix, was made, and immediate operation was performed under general anæsthesia.

Operation (*Fig.* 344).—The abdomen was opened by a subumbilical right paramedian incision. There was no free fluid. There were fine basal peri-appendicular adhesions, and the appendix was removed.

A cyst the size of a golf-ball was attached to the left pelvic wall. The presenting surface, about seven-eighths of the total area, was of a yellowish-white colour, the remainder being reddish at its pelvic attachment. The anatomical position of the cyst was comparable to that occupied by the left ovary in the female. It was retroperitoneal, attached by a base an inch in radius to the area just inferior to the bifurcation of the left common iliac vessels, and medial and inferior to the left ureter. Its medial relation was the rectum, and it was covered by coils of ileum, to neither of which had it any connexion, nor were these structures in any way abnormal.

A second cyst was present, inferior to the first, lying deep in the retroperitoneal tissues to the left of the rectum and above the left levator ani muscle.

The first cyst was aspirated, 50 c.c. of thick brown scintillating fluid being obtained; and the comment was made that there were probably cholesterol crystals in the fluid. The

cyst was then removed piecemeal as far as its attachment to the pelvic wall. The second cyst was aspirated, 40 c.c. of a similar fluid being withdrawn. No further treatment was considered advisable, having regard to the position of the cyst. There were no other cysts in the abdomen. Both kidneys, the ureters, and the urinary bladder were in their customary position, and in no way pathological. The remaining organs and viscera were healthy and in their normal position. There was no demonstrable connexion between the two cysts. A tube was placed near the remnants of the first cyst and the abdomen closed.

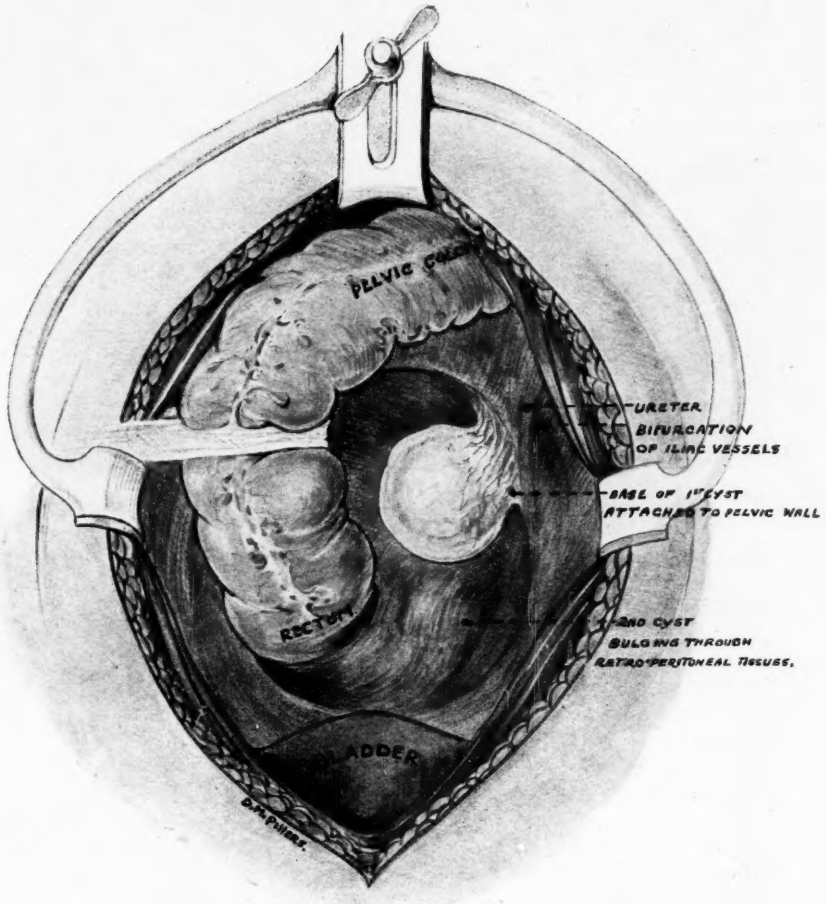


FIG. 344.—Showing the appearance of the cysts at operation and their relation to neighbouring structures.

PROGRESS.—The patient made an uninterrupted recovery and was discharged three weeks after admission. A rectal examination on the day of leaving showed that the lower cyst had refilled, but there was no discomfort, and defæcation was normal and not painful.

PATHOLOGICAL.—

The Contents of the Cyst.—A brownish, gravy-like fluid, with considerable numbers of red blood-cells, scanty pus cells, and large numbers of cholesterol crystals. No organisms seen on examining a smear. A culture gave no growth. Staining with Sudan 3 demonstrated the presence of fat particles.

The Cyst Wall.—The thickness of the cyst wall was 4 mm. at the base and 1 mm. at the free surface. In consistency it was firm—almost hard—and white in colour. The inside

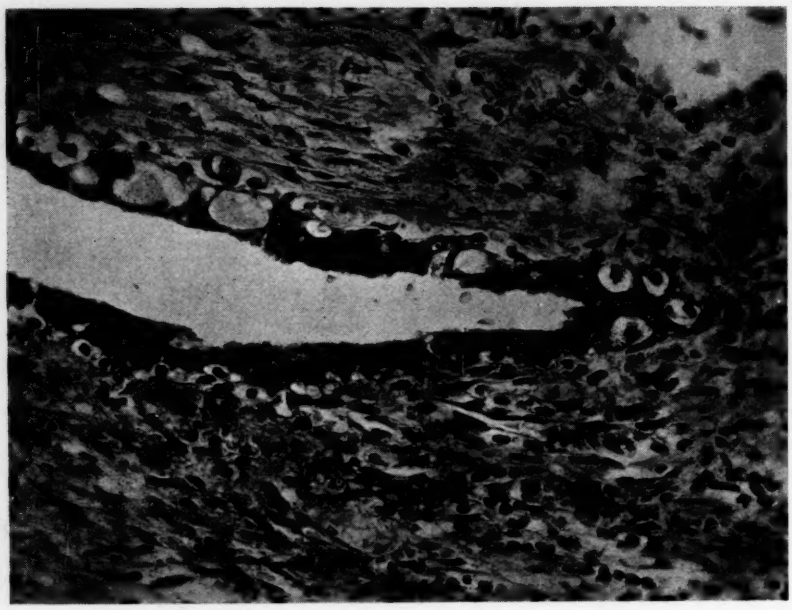


FIG. 346.—Microphotograph showing ciliated epithelium and large vacuolated cells in the epithelial lining. (× 300.)

FIG. 345.—Microphotograph showing the fibro-muscular construction of the cyst wall. (× 42.)

of the attached portion of the cyst showed areas of vascularity, with small islands of organized blood-clot. There were several diffuse hæmorrhagic areas, 1 mm. in diameter, over five-eighths of the free interior surface.

On microscopical examination (hæmotoxylin and eosin, van Gieson) a section from the free surface of the cyst showed a wall formed of dense fibrous tissue with few cells, and lined by indeterminate flattened cells.

Sections from the attached area showed a more cellular fibrous tissue wall, with a considerable amount of smooth muscle (*Fig.* 345). The epithelial lining consisted of a columnar ciliated epithelium, frequently flattened into cubical (*Fig.* 346). There were numerous small and large blood-vessels in the fibro-muscular tissue, with an occasional area containing small numbers of polymorphonuclear leucocytes. No secreting elements could be found.

FIG. 347.—Microphotograph of the appendix showing debris in the lumen and desquamation of the surface epithelium. (× 42.)

Appendix.—The lumen contained three fæcoliths. Microscopically, there was a considerable amount of cell debris in the lumen, with slight desquamation of the surface epithelium (*Fig.* 347).

Comment.—The pre-operative diagnosis of this case was " subacute pelvic peritonitis originating in the appendix ". The cause of the pelvic peritonitis was allotted to the appendix on account of the history and physical signs, and also because the appendix is the commonest cause of such a condition. In an older person the position of the mass on the left side of the pelvis, in association with the symptoms, would probably have led to a diagnosis of diverticulitis. The

correlation between the pre-operative right-sided abdominal signs and symptoms and the left-sided operative findings is difficult.

The cyst which was removed showed slight inflammatory changes, as did the appendix. The only logical explanation of these apparently contrary findings, is that the appendix was sufficiently pathological to cause the abdominal signs and symptoms, and the cyst sufficiently pathological to cause the diarrhœa and pelvic tenderness.

The cysts are classed as mesonephric in origin from the following considerations: (1) Their anatomical position and isolation from any adult viscus or organ; (2) The histological findings. There is a marked resemblance between the histological picture of these and parovarian cysts.

DISCUSSION

Before discussing the aetiology of mesonephric cysts and reviewing the literature, a brief account of the development of the human excretory system will be given.

Development of the Human Excretory System.—The main points in the development of the human excretory system are: (1) The parent structure is the intermediate cell mass. (2) Three sub-systems are developed from this: (*a*) The pronephros; (*b*) The mesonephros; (*c*) The metanephros.

a. The Pronephros.—Most of the pronephros atrophies, part—the primary excretory duct—persisting as the mesonephric (Wolffian) duct.

b. The Mesonephros.—The cephalic portion of the mesonephros atrophies; the caudal portion forms segments of the genital system.

c. The Metanephros.—This persists as the permanent adult kidney, and is composed of the caudal part of the intermediate cell mass and the ureteric diverticulum from the mesonephric duct. These facts have been summarized in the appended diagram (*Fig.* 348).

It will be noticed that the possibility for an embryological error to become manifest in the adult is confined developmentally to the caudal part of the mesonephros, mesonephric duct, and the junction between the metanephrogenic cap and the ureteric diverticulum.

Aetiology of Mesonephric Cysts.—The cysts represent persistent portions of the mesonephros and mesonephric duct. The numerous situations in which the cysts are found is dependent on the intricate development of the excretory organs.

The standard text-books on pathology contain practically no reference to the subject; one author, however, provides an explanation of the characteristics of the epithelial lining of retroperitoneal cystic tumours or enterocystoma, by saying that "many embryonal canals are originally lined by ciliated epithelium, as the neural, enteric, respiratory, and genital, and an early misplacement of cell groups from these structures would naturally have the lining cells in an embryonal state" (Ewing[17]).

As regards the number and histological appearance of the cysts, it may be said that unilocular cysts are derived from an aberrant or persistent mesonephric duct, and are unlikely to contain secreting elements, whereas multilocular cysts probably owe their origin to the mesonephros, and tend to have glomeruli and tubules in their wall.

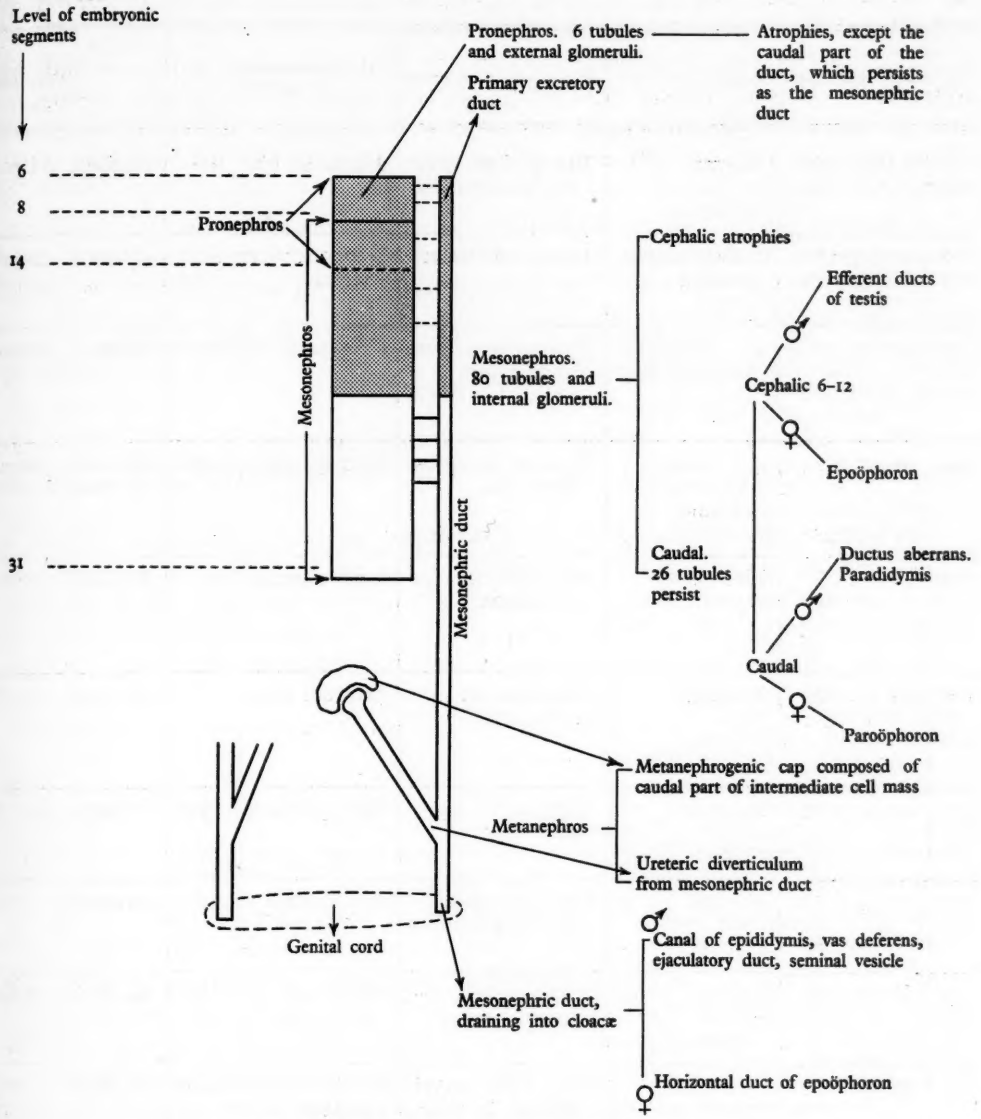


FIG. 348.—Diagram showing the development of the human excretory system and the adult homologues.

Review of the Literature.—In 1936 Hardeman[2] reviewed the recorded cases. Up to 1924, 44 cases had been recorded, but there was considerable confusion as to what constituted a "mesonephric cyst", and tumours of this variety were classified as "retroperitoneal", this heading affording protection to a heterogeneous collection of mesenteric, pancreatic, renal, and similar tumours.

Hardeman sieved the mass of material, and relieved the congestion considerably. He added 30 new cases, including 1 of his own, making a total of 74. Since then 5 fresh cases have been added, 1 each by Fiolle,[6] Gutiérrez,[13] Israel,[3] Costa,[10] and Peroni,[12] which, including the author's, makes a total of 80.

CASE NO.	AGE AND SEX	FAMILY HISTORY	SIGNS AND SYMPTOMS	PRE-OPERATIVE DIAGNOSIS	TREATMENT
1	2 yr. 3 mth. M.	Normal	Lump left loin. No genito-urinary	Dermoid cyst	Excised. Up in 8 d
2	2 yr. 2 mth. M.	1 sister a twin, normal	Lump abdomen	Tumour left iliac region	Excised. Dead in 7 d
3	41 yr. F.	4 children. Labours long	Prolapsed rectum since infant	Prolapsed rectum	Excised. Prolapse cu
4	36 yr. F.	4 children. Argentinian. Normal	Tumour and pain right loin	Retroperitoneal cyst	Excised. Recurrence 1 year
5	40 yr. F.	Normal	Pain left side for 2 months	Left ovarian cyst	Excised
6	32 yr. F.	Normal	Constipation for 6 months	Ovarian cyst	Excised
7	49 yr. M.	Normal	Tumour right loin	Retroperitoneal cyst	Excised
8	17 yr. M.	Normal	Cyst left epididymis for 8 months	Epididymal cyst	Excised
9	28 yr. M.	Normal	Tumour left inguinal region	Neoplasm (? endometrioma)	Excised + radium
10	36 yr. F.	Not given	Pain right sacral region 1 year. Tumour right hypogastrium	Posterior abdominal tumour	Excised
11	18 yr. M.	Normal	Pelvic peritonitis	Sub-acute pelvic peritonitis	1 cyst excised

Of these, 11 recent cases are here analysed in tabular form, the salient features of each case being recorded under general headings. These cases date back to 1924, and are to a certain extent selected, in so far that only those which contained sufficient data for the analysis have been considered, the literature on others being unavailable. The analysis, therefore, gives a fair representative average for this period.

ASES OF INTRAPELVIC CYSTS

Cysts		Associated Abnormalities	Histological
Position	No.		
etroperitoneal, anterior left idney, no pedicle	2	Not mentioned	Secreting elements. Fibro-muscular, smooth. Ciliated epithelium
etroperitoneal, post-abdominal all, attached to undescended esticle. Pedicle	15	Left agenesia. No left vas deferens or seminal vesicle. Right hydronephrosis	Secreting elements. Fibro-muscular, smooth. Flat epithelium
etroperitoneal, utero-vesical ouch. No pedicle	1	None	No secreting elements. Fibro-muscular, smooth. Ciliated epithelium. Supra-renal cortical rests
etroperitoneal, behind hepatic lexure, adherent to iliacus	1	None	No secreting elements. Ciliated epithelium. Adeno-carcinoma. Fibro-muscular, smooth
etroperitoneal attached to left ovary	Several	None	Secreting elements. Fibro-muscular, smooth. Cubical and ciliated epithelium. Papilliferous
etroperitoneal, hung from left ovary	1	None	No secreting elements. Cubical epithelium. Fibro-muscular, smooth. Malignant (?)
etroperitoneal, right loin. Uni-ocular	1	None	No secreting elements. Fibro-muscular, smooth. Indeterminate epithelium
perior pole left epididymis	1	None	No secreting elements. Ciliated epithelium. Fibro-muscular, smooth
umour present at 8 years, hought to be hernia. Operated on, but recurred	1	None, both testicles present	Secreting elements. Fibro-muscular, smooth. Malignant
etroperitoneal, behind cæcum. Unilocular	1	None	Cyst from remains of Wolffian duct
etroperitoneal, pelvis. No edicle. Unilocular	2	None	No secreting elements. Ciliated epithelium. Fibro-muscular, smooth

Certain facts emerge from this analysis, and together with the conclusions of previous writers, notably Maury[14] and Hardeman,[2] they are as follows :—

1. The tumours are confined almost exclusively to adults (2 recorded exceptions, *Cases* 1 and 2).

2. The incidence of malignancy is 50 to 60 per cent.

3. The tumours are cystic. They have a wall consisting of fibrous tissue,

smooth muscle, and vascular areas, and contain secreting elements (glomeruli and tubules). They are usually lined by ciliated epithelium.

4. They are retroperitoneal and have no direct continuity with an adult viscus or organ.

5. They are usually single and unilocular.

6. Hardeman[2] states that the female sex predominates, and according to Hinman[1] the balance is developmentally in favour of the female, because the pro-meso-metanephros complex is utilized in the male in forming the vasa deferentia and epididymes, whereas in the female the complex is vestigial, forming potential sources of cyst formation. It will be seen from the analysis, however, that 6 out of the 11 cases were of the male sex.

7. Inflammatory or mechanical changes are rare.

The absence of torsion is explained by the fact that most of the cysts have no pedicle. They seldom rupture, for not only are their walls thick, but also they are situated in a protected position on the posterior abdominal wall or in the pelvis.

Differential Diagnosis.—A primary diagnosis of mesonephric cyst is made very rarely, 2 cases only in the analysis (*Cases* 4 and 7) approaching a correct conclusion. The cysts are usually diagnosed as the commonest tumour in the situation they happen to be, such as ovarian, prostatic, and renal. No dogmatic criteria can be laid down for a correct pre-operative diagnosis. A more exact diagnosis should be made more frequently, however, as the cysts are fairly common. The failure to do so is apparently due to insufficient preliminary investigation.

Treatment.—This is surgical. The cysts should be removed completely, as there is a high percentage of malignant incidence.

Prognosis.—The prognosis in these cases is good. In the few instances where death occurs it is due to some complication, such as associated renal abnormalities, agenesia, hydronephrosis, etc. Even in cysts which are malignant, the malignancy is confined usually to the cyst itself, and in those cases where direct or metastatic spread has occurred, provided the tumour and the surrounding tissue is excised, the expectation of life compares favourably with any low-grade carcinoma elsewhere.

SUMMARY

1. A case is reported of two pelvic cysts in the male, of probably mesonephric origin.
2. The literature is reviewed, and some recent cases analysed.
3. The aetiology of mesonephric remnants is discussed.
4. The development of the human excretory system is outlined.

In conclusion the author would like to express his gratitude to Mr. A. J. Gardham for permission to publish the reported case, and for his valuable help and advice in the compiling of this paper; and to Dr. C. R. Lane for the histological preparations and reports. For the developmental particulars the author is indebted to Professor J. E. Frazer's *Manual of Embryology*,[15] and to a recent article by the same author.[16]

REFERENCES

1 HINMAN, GIBSON, and KUTZMANN, *Ann. of Surg.*, 1924, lxxix, 762.
2 HARDEMAN, D. R., and DE GROAT, A., *Ibid.*, 1936, ciii, 388.
3 ISRAEL, G. I., *Amer. Jour. Surg.*, 1936, xxxii, 369.
4 MIRIZZI, P. L., *Ann. d'Anat. pathol.*, 1935, xii, 945.
5 PORTES, H., and ISIDOR, P., *Ibid.*, 1934, xi, 528.
6 FIOLLE, J., *Bull. Soc. d'Obst. et de Gynécol.*, 1935, xxiv, 578.
7 GALINVAUX, *Rev. belge de Sci. méd.*, 1932, iv, 524.
8 PLOYE, M., *Jour. d'Urol.*, 1932, xxxiv, 292.
9 PICARD, E., and ESTAS, P., *Rev. belge de Sci. méd.*, 1930, ii, 689.
10 Author's case.
11 COSTA, A. J., and RONCHETTI, A. I., *Arch. argent. de enferm. d. ap. digest. y de la nutrición*, 1936, xi, 642.
12 PERONI, P., *Bollettino*, 1936, x, 193.
13 GUTIÉRREZ, A., *Boly. trab. de la Soc. de Cir. de Buenos Aires*, 1937, xxi, 298.
14 MAURY, J. M., *Surg. Gynecol. and Obst.*, 1918, xxvi, 663.
15 FRAZER, J. E., *Manual of Embryology*, 1931, 372.
16 FRAZER, J. E., *Jour. of Anat.*, 1935, lxix, 455.
17 EWING, J., *Neoplastic Diseases*, 3rd ed., 1928, 1033.

EPIDERMOIDS ARISING IN THE DIPLOE OF THE BONES OF THE SKULL*

By G. F. ROWBOTHAM, Manchester

An epidermoid is a neoplasm which arises in ectodermal cell rests and in particular from epidermal primordia. In comparison with other congenital cysts, the epidermoids must arise from the ectoderm at a very early embryological date, for the others, which are commoner, result from the failure of complete obliteration of the processes which form the face and the gill slits. This type is very apt to include dermal as well as epidermal elements, an important distinction. Dermoid cysts, as well as teratomas, occur, though rarely, inside the skull. It is, however, with the purely ectodermal tumours that this paper is concerned, and reports are given of the first two cases of epidermoids of the calvarium which have been recorded in this country.

Embryologically it is easy to visualize how ectodermal cell rests may become enclosed within the tables of the calvarial bones; both skin and brain arise originally in the same ectodermal cell plate and are later separated by the ingrowth of mesodermal elements from the lateral cell mass. The neoplasm now known as an epidermoid was originally described by the pathologists of the nineteenth century, and notably by Cruveilhier, who gave to it the most graphic name of *tumeur perlée*, which so exactly describes its glistening capsule. Johannes Müller (1838) coined the name 'cholesteatoma', an undesirable term, however, as it embraces more than one type of new formation—dermoids, epidermoids, and endotheliomas—as well as the non-neoplastic cheesy debris which forms in the mastoid air cells in chronic inflammatory processes.

A good deal of controversy has arisen on the problem of nomenclature, and it was Critchley and Ferguson (1928) who popularized the simple name of epidermoid, claiming that it was the most satisfactory as it implied the precise pathology which repeated histological evidence had established.

Mahoney (1936), in a long and comprehensive paper, reviewed all the cases (142) he could find in the literature, to which he added his own observations made on 5 cases collected in the laboratories of Professor Foerster, of Breslau. Müller (1836) was the first to describe an intradiploic epidermoid, and (apart from the temporal bone tumours, of which 6 cases have been described in a recent publication from this clinic) only 24 cases of epidermoids arising between the two tables of the skull have been described. Bucy recently described 3 cases, and was able to collect 13 only from the literature. It is doubtful whether the 2 examples attributed to Critchley and Ferguson by Mahoney should be included, particularly as the authors themselves said: "we met with no instance of a diploic tumour in our series".

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* From the Neurosurgical Clinic of the Manchester Royal Infirmary. This work was aided by a grant from the Dickinson Scholarship Fund.

CASE REPORTS

Case 1.—A swelling on the left side of the forehead, noticed for the first time three and a half years ago, had slowly increased in size, and was associated with a mild degree of atrophy in the left optic disc and faulty vision in that eye.

HISTORY.—A young man aged 26 was admitted to the Neurosurgical Service of the Manchester Royal Infirmary, under the care of Mr. Geoffrey Jefferson, on January 11, 1937. Ten years previously, at the age of 16, he contracted rheumatic fever and was kept in bed for eighteen weeks. He made a good recovery from this illness and was able to play games in the usual way. Six years ago he had an attack of influenza, which was complicated by the development of a corneal ulcer on the left eye. This ulcer healed completely, and even though no opacity or scar remained, the eyesight in that eye had never been completely regained. Three and a half years ago he was questioned about a swelling on the left side of his forehead, and from this time he noticed a slow but gradual increase in its size. Apart

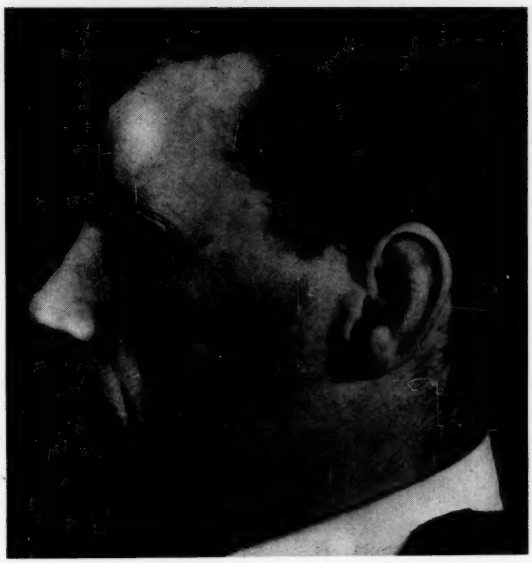


FIG. 349.—*Case* 1. Clinical photograph showing a small hemispherical swelling protruding from the left frontal bone.

from an occasional 'creepy' sensation in the overlying and neighbouring skin, the swelling was painless. In the past two and a half years he had several fainting attacks, in which everything seemed to go black for a moment or two and he felt as if he were going to fall. These attacks were followed by severe palpitations, and were referable to the cardiac muscle rather than to the brain.

ON EXAMINATION.—He was a well-developed young man of medium build, very intelligent and fully co-operative. There were no neurological signs apart from a slight pallor of the left optic disc, which was thought to be due to the effects of an influenzal papillitis, as no evidence could be found of an intracranial or chiasmal compression. On the left side of the forehead to its outer side and just above the eyebrow was a hemispherical swelling of rubbery consistency (*Fig.* 349). It measured 1¼ in. in diameter, and at its highest point stood about ½ in. above the surrounding tissues. It was not attached to skin, the superficial veins were not enlarged, and at parts of its periphery a hard sharp bony margin could be palpated. The Wassermann test was negative, the serum-calcium was 10·8 mg. per 100 c.c., the blood-count and chemistry of the cerebrospinal fluid were normal, as was the pressure of the cerebrospinal fluid measured on spinal puncture. It could therefore be concluded that an intradural extension of any importance was not present.

Radiography showed a defect in the left frontal bone immediately above the orbit and just to the inner side of the external angular process. It was roughly circular in shape, and measured about 1 in. vertically and 1 in. transversely. Its margins were clear cut and quite densely sclerosed, as was the neighbouring bone. This defect had obviously been caused by a pathological process of a benign nature. The rest of the skull was normal (*Fig.* 350).

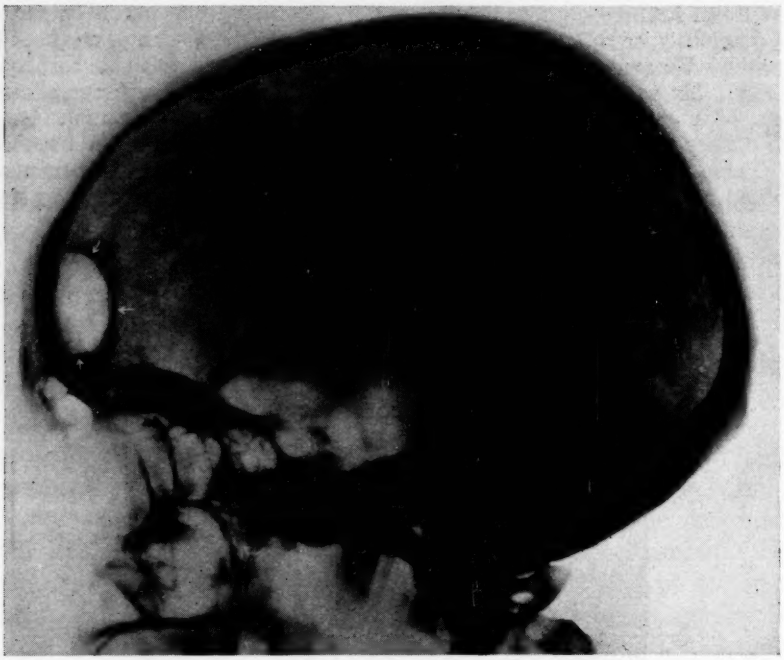


FIG. 350.—*Case* 1. Radiograph (lateral view) showing a circular defect in the left frontal bone. The edges are clearly defined, and there are sclerotic changes in the surrounding bone.

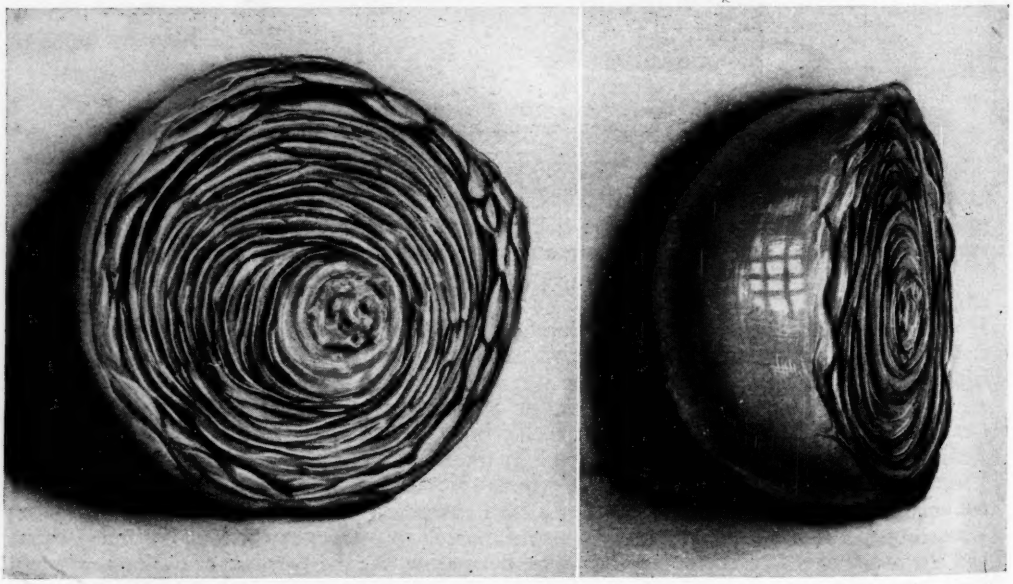


FIG. 351.—*Case* 1. A drawing by Miss Dorothy Davison of the epidermoid removed from the frontal bone, showing its laminated structure and the sheen on its capsule.

OPERATION, February 5, 1937 (G. F. R.).—A small Trotter's flap was turned down and the swelling found to be entirely beneath the pericranium. The galea moved freely over the tumour and there were no abnormal vessels running from the pericranium to the galea. On incising the pericranium at the upper periphery of the swelling and reflecting it downwards, a pearly white glistening membrane was seen, which was obviously the capsule of an

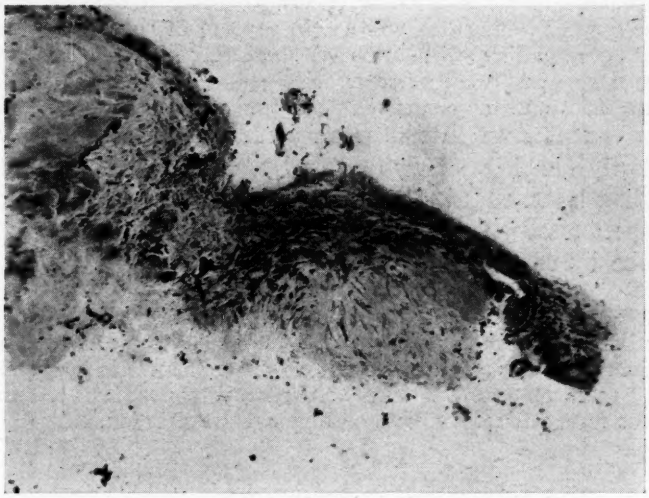


FIG. 352.—*Case* 1. Microphotograph of the under surface of the pericranium covering the epidermoid. It shows a proliferation of the fibroblastic cells which form part of the stratum durum. (Stain, hæmalum and eosin.) (× 80.)

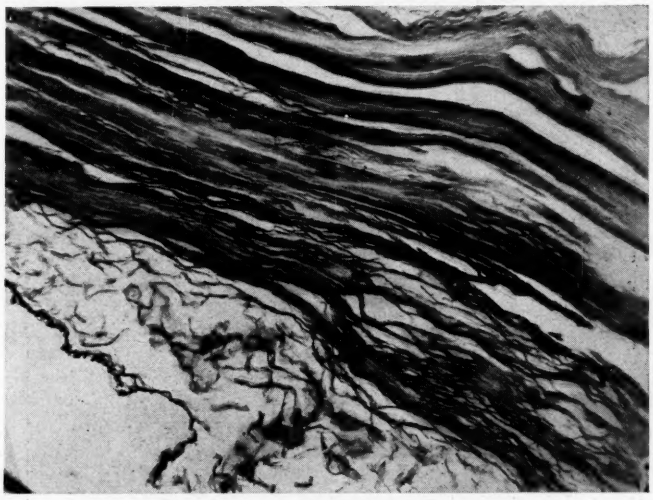


FIG. 353.—*Case* 1. Microphotograph of the laminæ forming the inner layer of the diploic epidermoid, showing the meshwork and parallel anuclear fibres which correspond with the stratum corneum in the skin. Many of the spaces were originally filled with cholesterol crystals. (Stain, hæmalum and eosin.) (× 80.)

epidermoid. The outer table of the bone had entirely disappeared, the edges of the bone were smooth and raised like the mouth of a crater, and after the tumour had been shelled out the inner table was found to be firm and had not been perforated. The cavity was carefully curetted and treated with Zenker's fluid in order to destroy any possible remaining capsule. Pieces of bone were nibbled away from the edges for microscopical section.

PATHOLOGY OF THE SPECIMEN.—

Macroscopical (*Fig.* 351).—The tumour looked like a glistening white ball, and transverse section showed it to be composed of thick concentrically disposed laminæ.

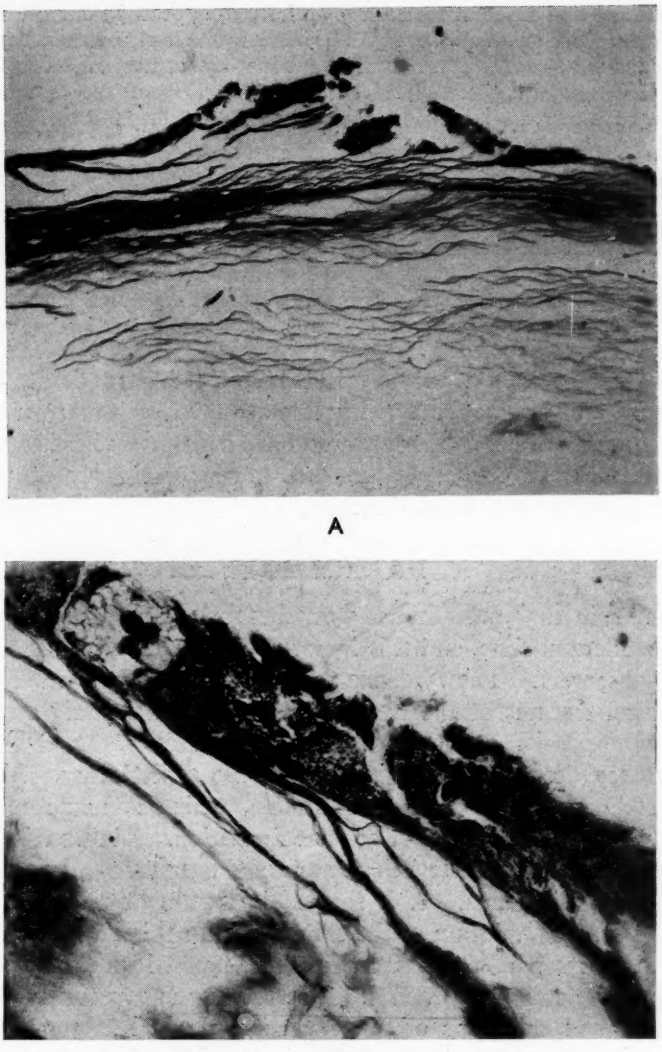
Histological (*Figs.* 352, 353).—The tumour was fixed in 10 per cent formol saline, embedded in paraffin, and sections were stained with hæmalum and eosin, phosphotungstic acid, and van Gieson stains. The epidermoid was composed of several coats. The outermost covering consisted of a fine acellular connective tissue containing small blood-vessels towards its inner aspect; lining this was a thin stratum of stratified epithelium, and this layer enclosed long parallel fibres without nuclei, alternating with a fine meshwork of anuclear tissue. No dermal elements were present. The epithelial layer represented the stratum granulosum of the skin, and the central acellular fibres the stratum corneum.

Comment.—This case illustrates very clearly the life history of a diploic epidermoid—slowly growing, slowly destructive, and excitatory to sclerotic reaction in the surrounding bone. Had this tumour been allowed to grow further it would probably have perforated the inner table and caused embarrassment of the underlying frontal lobe by compression.

Case 2.—E. P., a woman aged 35, was admitted to the Neurosurgical Service of the Manchester Royal Infirmary on January 1, 1937, under the care of Mr. Geoffrey Jefferson. Six months previously she had a convulsive seizure whilst she was in bed, and from that time the chronology of the symptoms had been headaches, dizziness, further seizures starting in the left side of the face, failure of vision, and mental deterioration.

FIG. 354.—*Case* 2. Radiograph (antero-posterior view) showing an erosion in the left occipital bone, chiefly below but extending above the superior curved line. It is roughly oval in shape, with its long axis vertical. Its margins are crenated and the edges of the bone sclerosed. This is a typical picture of a benign destruction of bone.

ON EXAMINATION.—Apart from a little unsteadiness in walking, all the major neurological signs were referable to the lower part of the motor cortex in the right cerebral hemisphere. There was no visible or palpable swelling on the skull. Radiography showed a defect in the left occipital bone. It was roughly oval in shape, with crenated margins, and measured $1\frac{3}{8}$ in. in its vertical axis and $1\frac{1}{4}$ in. transversely. It was situated chiefly below the



A

B

FIG. 355.—*Case* 2. Microphotographs showing the stratum corneum lined with a layer of degenerating cells filled wih granules, probably of a cholesterol nature. A, Low-power view (× 80); B, High-power view (× 400). (Stain, hæmalum and eosin.)

superior curved line, but extended above it for a distance of $\frac{1}{2}$ in. The bone at the edge of the defect was sclerosed, indicating the benignity of the lesion, and the rest of the skull was normal. (*Fig.* 354.)

In this case there were neurological signs pointing strongly to a lesion in the left cerebral hemisphere, and radiographical evidence of pathological changes in the posterior fossa. Two entirely separate conditions were found—an epidermoid in the occipital bone and a diffuse astrocytoma in the right temporal lobe of the brain, to which the symptoms and neurological signs were entirely attributable.

OPERATION, January 13, 1937 (G. F. R.).—On a diploic epidermoid of the left occipital bone.

A transverse incision was made along the superior curved line of the occipital bone on the left side in its outer half. As the galea was incised and retracted upwards and the occipital muscles swept downwards, a white glistening membrane was seen projecting slightly from a defect in the occipital bone which straddled the superior curved line. The tumour separated easily from the bone and the defect was seen to be a vertical oval with crenated edges. Both external and internal tables had entirely disappeared, and the tumour capsule was resting on the underlying dura mater. The dura mater itself had not been invaded, nor had it been separated from the surrounding bone by an extension of the tumorous material into the neighbouring extradural spaces. All traces of the tumour capsule were removed and the edge of the bony defect was excised by nibbling forceps.

PATHOLOGY OF SPECIMEN.—

Macroscopical.—The capsule of the epidermoid was white and shiny like mother of pearl, and cross section showed it to be composed of thick white concentric laminæ.

Microscopical (*Fig.* 355).—It was difficult in this case to procure satisfactory sections of the epithelial cell layer because it was so friable and tore readily from the deeper structures. The deeper layers showed the typical long parallel fibres without nuclei enclosed in a fine meshwork of anuclear material which corresponds with the stratum corneum of the skin. Here and there could be seen degenerated cells filled with heavily staining granules, no doubt of a cholesterol nature.

Comment.—This was a very typical type of diploic epidermoid. It had eroded both tables but was not sufficiently large to give rise to any serious compression of the brain, though this certainly would have happened as the tumour increased in size. Epidermoids in our experience are not uncommonly found along with neoplasms of a different pathological nature.

PATHOLOGY

It was Remak, in 1854, who first suggested that the pearly tumour of Cruveilhier originated in ectodermal rests, and this hypothesis has been very strongly supported by subsequent observers. The evidence for this hypothesis, however, rests entirely on the histological features of the tumour itself, and particularly on the details of the capsule.

Percival Bailey described several layers in the typical case. Most externally is the stratum durum or glistening membrane, which is made up of layers of collagen fibres, with little evidence of cellular formation. Lining this is the stratum granulosum, which is the characteristic feature of the tumour and on which Remak's hypothesis essentially depends. It is formed by layers of epithelial cells such as are found in the area granulosum of the skin. Within this the tumour is composed of layers of concentric parallel anuclear fibres enclosed in a fine meshwork which corresponds with the stratum corneum. The tumour is avascular. That the final histological picture of this type of neoplasm is epithelial in structure is without question, and that it is epidermal seems certain, as the typical case does not contain dermal structures such as glands or hair.

The explanation why one ectodermal cell develops into skin and one into neural tissue has recently been investigated by workers in the Strangeways Research Laboratories at Cambridge, who believe that an ectoderm cell in its very earliest stages is not predestined to become either skin or neural tissue, but that its destiny is decided by the influence of the neighbouring mesoblastic tissue. To prove this, a piece of ectoderm, which, from its position in the embryo, is known to be developing into skin, if grafted into suitable surroundings can be made to differentiate into neural tissue. The factors which decide the type of differentiation are under what

is known as ' organizer influence '. Jefferson believes that the epidermoids found in the brain substance or within the membranes possibly are due to the failure of the organizer influence to compel groups of ectodermal cells towards neurogenic differentiation, and that they do not necessarily arise in rests which are predestined to form skin. However, as regards the diploic epidermoids, it is just as easy to

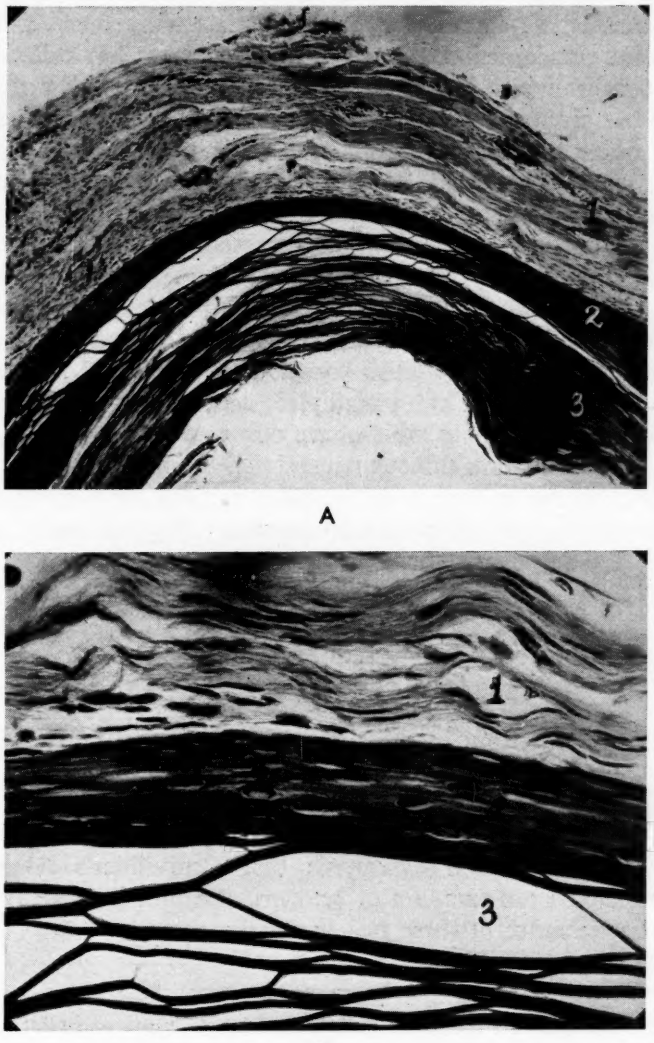


FIG. 356.—Microphotographs of an implantation epidermoid. The three layers can be clearly seen: 1, Stratum durum; 2, Epithelial layer or stratum granulosum, which characterizes these tumours; 3, Stratum corneum, showing the long anuclear fibres. A, Low-power view (× 55); B, High-power view (× 320). (Stain, hæmalum and eosin.)

visualize the misplacement of cell rests as to invoke the speculation of faulty organizer influences. What is entirely speculative of course is the essential origin of these tumours. Do they arise in ectodermal cell rests or do they develop through the phenomena of metaplasia from endothelial elements. As far as the author is aware, no epithelial rests have ever been discovered within the diploe of the bones

of the skull, and this has been his own experience in the examination of sections taken at random from over 100 skulls in an investigation carried out in the hope of finding abnormal cell rests which would explain the origin of some of the osteomas. This is a negative point, but it is important in that it shows that cell rests have been searched for and that one day they may be found.

In clinical pathology Nature has made many interesting experiments in the production of traumatic epidermoids, which illustrate with some certainty that they can develop from ectodermal cell rests. Professor Baker has collected a series of implantation tumours which have been produced by injuries of various kinds, and very kindly he has given me the opportunity to examine their histological structure. One of these, which grew as the result of a pig bite, was indistinguishable from a true developmental epidermoid. It occurred as a small rounded tumour, 5 mm. in diameter, embedded in the soft tissues of the finger. The outer coat consisted of wavy fibres of collagen material which corresponds with the nacreous capsule or stratum durum of Bailey; this was lined by a layer of flattened epithelium ten to fifteen cells deep, which was similar to the stratum granulosum of the skin and exactly like the epithelial layer of a typical epidermoid. The centre of the tumour was filled with the long thick, whitish concentrically disposed anuclear fibres seen in the stratum corneum of the two intradiploic epidermoids described in this paper and in the intracranial examples which have occurred in this clinic (*Fig.* 356).

The type of implantation tumour depends on the nature of the tissue which is inoculated, and if dermal as well as epidermal structures are carried into the deeper tissues and survive, a true dermoid will develop. Experimentally, grafts of skin implanted into the deeper structures usually grow for a time and then die and become purely cystic instead of neoplastic, and probably it is the death of a cell rest which accounts for so many implantation tumours being found as simple thin-walled cysts and not as solid tumours.

SUMMARY

Two epidermoids arising in the diploe of the frontal and occipital bones of the skull are described. In neither case did the neoplasm cause compression of the brain—in the frontal bone the outer table only had entirely disappeared. To prevent recurrence, a complete removal or destruction of the epithelial capsule is essential. Remak's original hypothesis that Cruveilhier's *tumeur perlée* and Müller's cholesteatoma are tumours of ectodermal origin is strongly supported by the histological appearance of these two personal cases.

REFERENCES

BAILEY, P., " Cruveilhier's Tumeurs Perlées ", *Surg. Gynecol. and Obst.*, 1920, xxxi, 390.

BUCY, PAUL C., " Intradiploic Epidermoid (Cholesteatoma) of the Skull ", *Arch. of Surg.*, 1935, xxxi, 190.

CRITCHLEY, MACDONALD, and FERGUSON, FERGUS R., " The Cerebrospinal Epidermoids (Cholesteatomata) ", *Brain*, 1928, li, 334.

CRUVEILHIER, J., *Anatomie pathologique du Corps humain*, 1829–35, Bk. 1, Liv. 2, Pt. 6.

JEFFERSON, G., and SMALLEY, A. ASHTON, " Progressive Facial Palsy Produced by Intratemporal Epidermoids ", *Jour. Larygnol. and Otol.*, 1938, liii, 417.

MALONEY, W., " Die Epidermoide des Zentralnervensystems ", *Zeits. f. d. g. Neurol. u. Psychiat.*, 1936, clv, No. 3, 416.

MÜLLER, JOHANNES, " Ueber den feinern Bau und die Formen der krankhaften Geschwülste ", 1838. Berlin: G. Reimer.

REMAK, R., " Ein Beitrag zur Entwickelungsgeschichte der krebshaften Geschwülste ", *Deut. Klin.*, 1854, vi, 170.

WADDINGTON, C. H., *How Animals Develop*, 1935. London: George Allen & Unwin.

OBTURATOR HERNIA

ITS AETIOLOGY, INCIDENCE, AND TREATMENT, WITH TWO PERSONAL OPERATIVE CASES

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Obturator hernia, or thyroidal hernia, may be defined as one which passes through the obturator foramen in the os innominatum. It is not a very rare variety of hernia, as over 400 cases have been recorded. In comparatively recent years only has operative treatment for this form of hernia proved successful. This is partly due to the fact that nowadays the condition is more frequently diagnosed before strangulation takes place than it was formerly. During the last century Hilton, Coulson, and Godlee all operated upon patients with strangulated obturator herniæ, but none of them succeeded in obtaining a successful issue.

The condition, which was discovered in an old female dissecting-room subject aged 68, first aroused my interest in 1912. The subject was stated to have died of bronchitis and cardiac failure. When the abdomen was opened and the abdominal cavity examined, an obdurator hernia was discovered, the hernial sac containing a Meckel's diverticulum which arose from the ileum just 24 in. from the ileocæcal valve. The diverticulum, just over 3 in. long from base to apex, had completely passed through the obturator foramen (*Fig.* 357). The lumen of the ileum in the vicinity of the diverticulum was not obstructed, but there were definite signs of former inflammation in the diverticulum itself, and it could not be withdrawn from the peritoneal sac. On dissecting the hernia from in front, a small sac was found under cover of the pectineus and obturator externus muscles. The obturator artery was situated behind the neck of the sac on its outer side, while the obturator nerve was found just above the artery. The anterior branch of the obturator nerve was lying immediately in front of the sac and must have been subjected to some pressure. No other abnormalities were found in the abdomen. It was the discovery of this hernia which stimulated my interest in the obturator region, and I subsequently found quite a number of small obturator hernial sacs, but without contents, in dissecting-room and post-mortem-room subjects.

Fig. 357.—Drawing made from dissecting-room subject. A Meckel's diverticulum in an obturator hernial sac.

CASE REPORTS

Case 1.—Mrs. Clara B., aged 65, was admitted to hospital in May, 1922, with acute abdominal pain of two days' duration. The abdomen was rigid, and a tumour, which

appeared to be about the size of a fœtal head, could be felt rising out of the pelvis on the left side. Vaginal examination was painful and caused a painful sensation down the thigh as far as the knee; the significance of this, however, was not appreciated at the time. The patient was a fat subject and was habitually constipated. On the day of admission she vomited twice. The temperature was 99° F., pulse 100, and respiration 22. The diagnosis appeared to be a twisted ovarian cyst.

AT OPERATION.—Three hours after admission, the abdomen was opened through a left paramedian subumbilical incision. A solid left ovarian dermoid tumour was found, with a twisted pedicle. The pedicle was transfixed and the tumour removed. On inspecting the pelvic cavity prior to closing the wound a knuckle of ileum was found to be herniated through the left obturator foramen; the whole circumference of the gut was not included in the hernial sac, so that the hernia was of the Richter type. As the herniated portion of bowel could not be reduced from the sac by simple traction, my assistant made gentle pressure on the sac from the groin while I employed gentle traction from above, and the hernia was eventually reduced. The next step was to invert the sac, and this proved a matter of some little difficulty. However, it was eventually accomplished by seizing the fundus of the sac with clip forceps and making firm but steady traction. The neck of the sac was then transfixed with a silk suture and the redundant sac removed. The stump of the sac was covered over with sutures, which flattened it out against the obturator foramen. The abdomen was then closed in layers.

Recovery was uneventful, and when seen a year later the patient was in good health.

Case 2.—Mrs. George H., aged 58, was admitted to hospital with acute intestinal obstruction on April 4, 1937. The patient was the mother of eleven children, nine of whom were alive and well. She had always enjoyed good health, with the exception of recurrent attacks of bronchitis every winter. For some two years prior to admission she was conscious

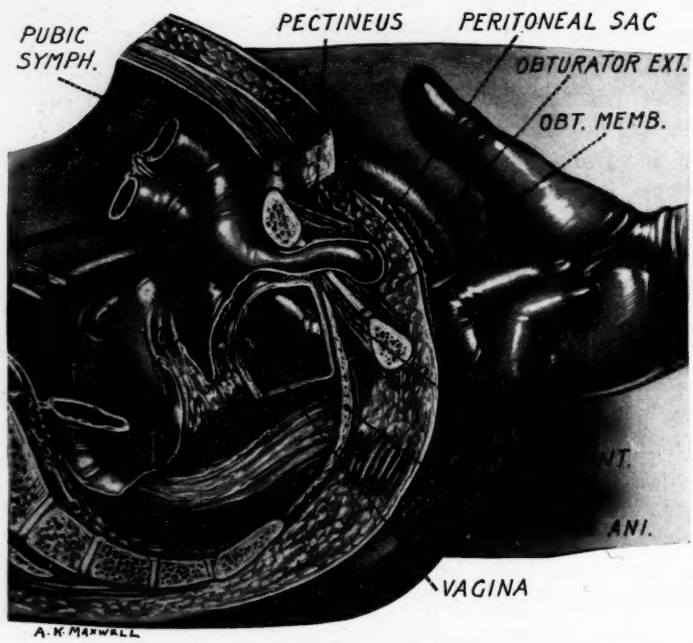

FIG. 358.—Drawing showing the relations of an obturator hernia and how a vaginal examination may aid diagnosis.

of pain of an intermittent character in the left groin. She had also been receiving treatment from her panel doctor for pain in the left knee, which was stated to be rheumatic in nature.

Two days before admission the patient was not feeling very well, having been in bed for a week with bronchitis. She was very constipated and was given castor oil in order to

evacuate her bowels. Some three hours later she was awakened by acute abdominal pain and vomiting, which persisted for several hours; her doctor was called in and immediately sent her into hospital.

ON EXAMINATION.—The patient was found to have a distended, tender, and somewhat rigid abdomen. There was a slight bulge in the left obturator region, and on flexing the thigh and rotating it outwards in order to relax the muscles, a small tumour about the size of a large cherry could be felt. It was tender, tense, and irreducible. There was no impulse on coughing, the tumour was dull on percussion, and pain was experienced in the left knee-joint. On making a vaginal examination a tender mass could be felt in the region of the left obturator foramen (*Fig.* 358). A strangulated obturator hernia was diagnosed, and operation was decided upon.

AT OPERATION.—Under gas and oxygen anæsthesia the abdomen was opened by a left subumbilical paramedian incision. The small intestine was distended, and it was difficult to examine the contents of the abdomen; however, by means of hot saline towels the lower

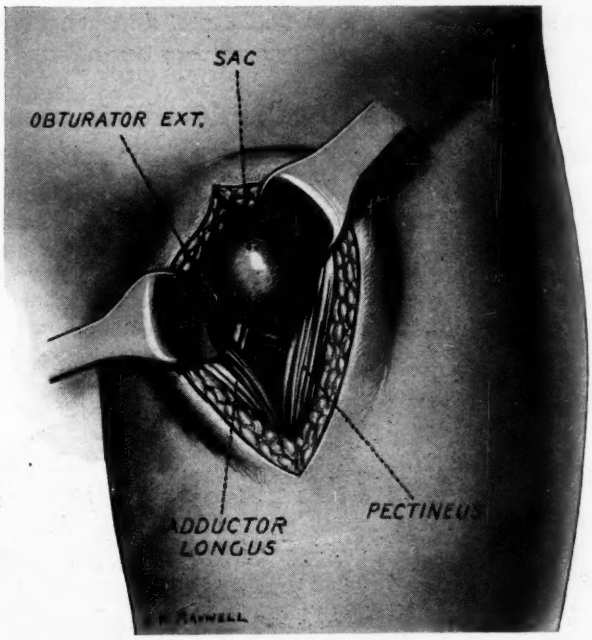

FIG. 359.—*Case* 2. Obturator hernia exposed from in front.

abdomen was eventually explored and a loop of the terminal ileum was found strangulated in the obturator canal. All attempts to reduce the hernia from the abdominal aspect having failed, the abdomen was covered with hot towels and the left obturator region explored. The left thigh was flexed and abducted and a vertical incision was made over the most prominent part of the tumour. The saphenous vein was divided between two ligatures and the tense sac of the hernia was exposed by retracting the pectineus muscle outwards, and the adductor longus muscle inwards (*Fig.* 359). The obturator nerve was found on the inner side of the sac, lying on the obturator externus muscle. The sac was opened at the fundus and an acutely inflamed knuckle of small bowel was exposed. As the bowel could not be reduced, a finger was carefully passed up lateral to the sac and insinuated into the obturator canal, thereby stretching the tissues in the vicinity. After this procedure the bowel was reduced from the abdominal aspect. The hernial sac was then dissected out and transfixed at its neck and excised. As very definite weakness in the region of the obturator canal was then found, a strip of the innermost fibres of the pectineus muscle was raised and stitched to the periosteum above the obturator canal. The wound was then closed and covered with a dressing.

The abdomen was now inspected again and the loop of gut in which the hernia had occurred was lifted out of the pelvis and carefully examined. The herniated portion consisted

of a knuckle in which nearly the whole circumference of the bowel was involved. It was covered with hot saline towels; after five minutes it was decided that the bowel was viable and it was returned to the abdomen. As the patient's condition was rather poor, a pint of 6 per cent glucose saline was given. The abdominal wall was closed in layers and a tight many-tail bandage applied. A tight spica bandage was applied over the dressing in the left groin.

The patient was kept in the operating theatre for one hour after the operation, during which time her condition improved considerably. On return to the ward 5 oz. of black coffee were given per rectum. With the exception of some bronchitis, which necessitated a stimulant, recovery was rapid and the patient left hospital three weeks after her operation.

Comment.—The interesting feature in this case was the time interval between strangulation and operation, practically 48 hours, which is apparently a very long time; but on consulting the literature on the subject I was surprised to find that the average duration of strangulation was six days, operation or taxis being used successfully in some cases and death occurring in others. Watson computed the duration of strangulation in 171 recorded cases, and concluded that the average was six days before an operation was performed or death took place.

MUSEUM SPECIMENS

The following three specimens in the Museum of the Royal College of Surgeons are of interest as they are the only three to be found in the Museum, and a minute examination of them does throw some light on the anatomy of the condition.

Specimen R.C.S. 6787.1.—*Fig.* 360 illustrates a portion of the right hip-bone, with the adjacent soft parts dissected in order to show the sac of an obturator hernia. This specimen was removed post mortem from a woman, aged 64, who was admitted to St. Marylebone Infirmary for abdominal pain, vomiting, and constipation of four days' duration. There

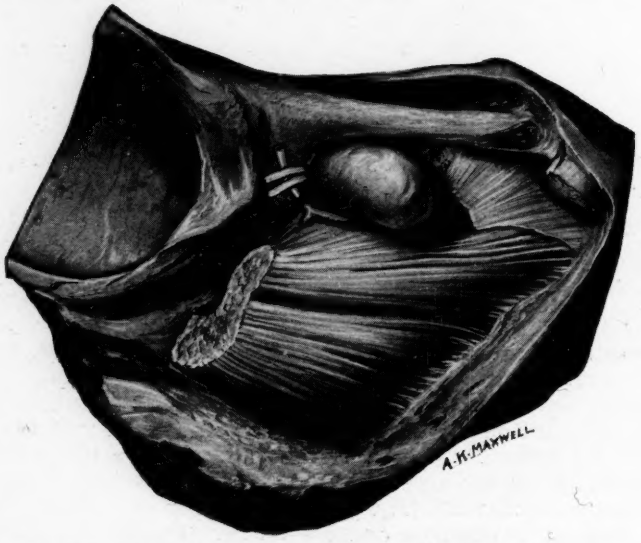

FIG. 360.—Obturator hernia (Specimen 6787.1 in R.C.S. Museum).

was no physical sign of hernia. Death occurred 42 hours after admission, and the autopsy revealed a hernia of the ileum into the right obturator canal. The specimen was sent to the R.C.S. Museum, and Mr. Targett reported on it as follows:—

"The portion of bowel obstructed consists of a coil of small intestine, but of this merely a small knuckle, not more than an inch in length, was strangulated. The ring of the constriction showed that the entire calibre was not included (Richter's hernia). The strangulation, however, was very severe, for nearly half the knuckle was in a sloughing condition. It

is probable also that there was a perforation at the apex of the knuckle at the time of death. The bowel above the obstruction was congested and distinctly dilated.

"Dissection of the sac showed that it had passed through the canal in the right obturator foramen (*Fig.* 360). The extreme length of the sac was 2 in.; the diameter of its mouth was not more than ¼ in.; and on reflecting the peritoneum it was seen that the lower and inner borders of the ring through which the sac passed was formed by a thin but very dense edge of fascia (obturator) lining the obturator internus muscle. The obturator vessels and nerves approached the neck of the sac from the outer side, and then parted company. The obturator artery gave off its pubic branch (which was of considerable size) before entering the ring. The pubic branch passed immediately below the neck of the hernia to the back of the pubes. The main trunk of the obturator artery entered the ring, and then passed obliquely across the obturator canal immediately below the hernial sac. In front, it divided upon the anterior surface of the thyroid membrane into its internal and external branches. The obturator vein accompanied it, but was remarkable for having a communication with a much larger vein lying upon the superficial surface of the obturator externus muscle. The obturator nerve and its branches, as seen from in front, were lying to the outer side of the hernia. The hernial sac was reduced into the pelvic cavity with difficulty owing to the obliquity of the canal and the small size of its deep (pelvic) orifice. The deep or posterior wall of the canal in which the hernia had been situated was formed by: (1) The obturator fascia and origin of the obturator internus muscle for half an inch or so; (2) An edge of obturator membrane; (3) The thick upper margin of the obturator externus muscle. The extreme length of this deep wall of the canal was 1½ in.

"When dissecting the hernia from in front it was found that the sac was covered with a moderately thick layer of subperitoneal fat, and outside this with a thin but distinct fascial sheath. The formation of the sac caused the peritoneum on the right wall of the bladder to be drawn upon, and consequently the bladder was pulled over to the right wall of the pelvic cavity. When put on the stretch, the peritoneum between the right wall of the bladder and the neck of the sac was less than ¾ in. in length. The bladder was fasciculated, and presented two moderately large sacculi, which were situated a little in front of the orifices of the ureters."

The clinical notes of this interesting case were reported by Sinigar in the *British Medical Journal* in 1897.

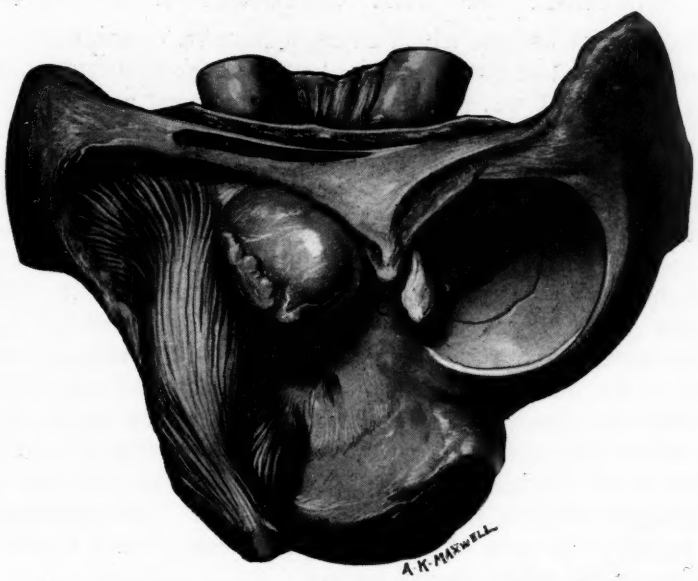

FIG. 361.—Obturator hernia (Specimen 6788.1 in R.C.S. Museum).

Specimen R.C.S. 6788.1.—*Fig.* 361 depicts part of the left hip-bone from a woman aged 78. The obturator externus muscle has been reflected in order to expose the obturator

membrane and an obturator hernia about the size of a walnut which projects through the obturator canal. The sac contains a knuckle of small intestine, apparently including a part of its circumference only.

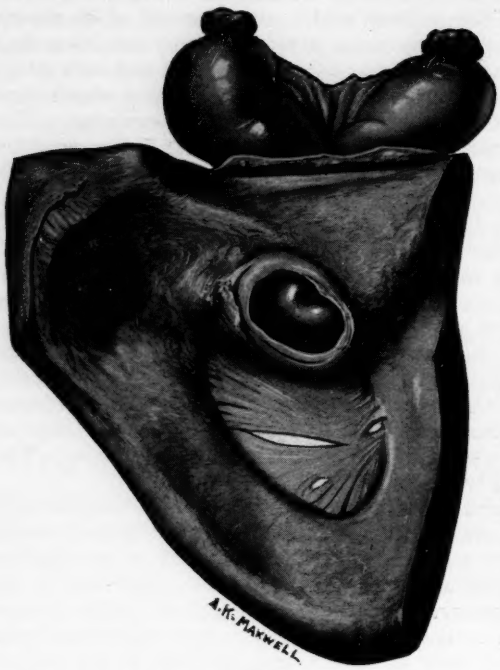

FIG. 362.—Obturator hernia (Specimen 6789.1 in R.C.S. Museum).

The clinical history of this case was reported by King in 1842. A brief summary may be appended here: The patient was a woman aged 78 who had complained for a year of pain in the left groin, in the course of the obturator nerve, and of cramp in the left thigh and leg. About a fortnight before her death she was seized with pain in the lower part of the abdomen, vomiting, and total stoppage of the bowels. Death occurred from intestinal obstruction and peritonitis. The herniated portion of the intestine was strangulated and beginning to slough. The omentum was adherent to the side of the bladder and left brim of the pelvis.

Specimen R.C.S. 6789.1.—*Fig.* 362 illustrates an interesting specimen of part of a pelvis, with a strangulated hernia of a small knuckle of ileum through the obturator canal on the left side. The anterior wall of the sac has been removed; the lower border of its neck is bounded by the margin of the obturator membrane. There are no clinical notes regarding this speciment.

ANATOMY OF THE OBTURATOR REGION

The first point of interest which emerges from an examination of subjects in the dissecting-room and at autopsy is the length of the obturator canal. In some fifty subjects examined I was surprised to find the canal was considerably longer than I had anticipated. Its average length in the fifty subjects was $1\frac{1}{4}$ in. The obturator canal is a fibro-osseous structure which is situated above the upper border of the obturator membrane. The canal itself is in reality larger than one would imagine; it just admits the index finger, but its walls are firm and unyielding, and it is easy to see how strangulation is brought about. The upper boundary of the canal is formed by the obturator groove on the inferior surface of the superior pubic ramus. The lower boundary is formed by the junction of the parietal pelvic fascia with the upper border of the obturator membrane over the upper border of the obturator internus muscle. The canal runs obliquely downwards, forwards, and inwards, and transmits the obturator vessels and nerve, the nerve lying above the artery. On examining the peritoneum which covers the internal orifice of the canal, it is found, as a rule, to be quite movable and is usually clothed by an appreciable amount of subperitoneal fat. This subperitoneal fat may play a part in the production of a hernia in this region much in the same way as it does in some of the other external herniæ.

The obturator membrane is really an aponeurosis of fibrous tissue, the fibres of which are arranged in an irregular, decussating manner; it cannot be said that there is any very constant feature regarding its structure. The membrane is

attached to the posterior margin of the circumference of the obturator foramen, except superficially opposite the obturator groove. The obturator internus muscle covers its posterior or pelvic surface, while the obturator externus muscle covers its anterior or femoral surface.

There is a very definite superficial part of the obturator membrane which can be made out quite easily in most dissections and may be termed the 'external obturator membrane'. This superficial part arises from the transverse ligament of the acetabulum, and runs almost horizontally inwards to the ramus of the pubis (*Fig.* 363). This superficial part of the obturator membrane is of some considerable importance, as it has a bearing on the course taken by some obturator herniæ.

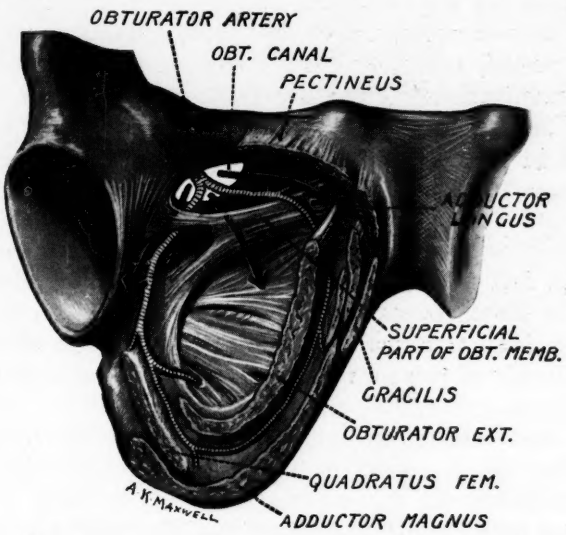

FIG. 363.—The anatomy of the obturator canal and membrane.

For the sake of completeness obturator herniæ may be classified into three varieties, as suggested by Picque and Poirier:—

1. In the obturator canal. This is by far the most frequent type. The sac of the hernia passes through the obturator canal and emerges under cover of the pectineus muscle.

2. Between the middle and superior fasciculi of the obturator externus muscle. The course of the sac in this type is along the route taken by the inferior branches of the obturator artery and nerve, the fundus of the sac passing between the superior and middle fasciculi of the obturator externus muscle.

3. Between the external and the internal obturator membrane. This is the rarest form of hernia, the sac passing downwards and forwards between the internal and external (superficial) obturator membranes (*Fig.* 363). If such a case is encountered the sac cannot be freed until the superficial part of the obturator membrane is incised.

THE AETIOLOGY OF OBTURATOR HERNIA

As one would expect, obturator hernia is much more common in females than males. Although the obturator foramen is smaller and more triangular in women, yet its transverse diameter is greater than in men.

Pregnancy is an important factor in the causation of obturator hernia, because not only does it cause a great increase in intra-abdominal pressure, and so throw a strain on all the hernial orifices, but after repeated pregnancies the parietal peritoneum is in a very relaxed condition. As to the relative frequency in the two sexes, this is a difficult matter to estimate with exactitude. Pimbet collected 73 cases in which there were only 8 men (or 10·95 per cent), while Watson found that out of 288 recorded cases 44 were men (15·27 per cent). The actual figure probably lies somewhere between 10 and 15 per cent for males.

The age incidence is for the most part between 60 and 70, although a few cases have been reported where the patient was as young as 12 years of age. The fact that the common age is somewhat over 60 years is important, because another factor comes into play—namely, the chronic chest conditions which so commonly occur at this age period, especially chronic bronchitis and emphysema. Also, in the aged female constipation is often prevalent, and there can be no doubt that constant coughing and straining at stool do act as predisposing causes. That this is so is illustrated in some cases where multiple herniæ are present in the same individual.

SYMPTOMATOLOGY

It is a curious fact that the signs and symptoms of an obturator hernia are very varied, and in those cases where there is no palpable tumour the condition is rarely diagnosed until intestinal symptoms occur. Even when a small hernia is present, it is difficult to palpate the swelling, as the sac is situated on the deep aspect of the pectineus and adductor longus muscles.

Probably the most constant symptom is pressure on the obturator nerve (*Fig.* 364), and even when this is present it is likely to be overlooked and the referred pain in the knee mistaken for rheumatic arthritis of the joint, as the condition is frequently associated with chronic arthritis of the joint, especially in elderly females.

Referred pain from the obturator nerve is a marked feature in cases of strangulated obturator hernia, even when gangrene of the gut has supervened.

It is most important to make a very careful and thorough examination of all the hernial orifices in cases of vague abdominal pains and discomfort, as it is only by so doing that a case of obturator hernia may be discovered.

It is more than likely that a partial enterocele occurs in not a few cases of obturator hernia, thereby casuing intermittent pain not only in the abdomen but also in the thigh muscles; in such cases a vaginal examination may be most useful in clinching the diagnosis in a female. In quite a number of cases the diagnosis can only be made by opening the abdomen, when the cause of the vague symptoms will become manifest. Nevertheless, the surgeon, although he cannot expect to meet with many cases of this type of hernia, should be conscious of the possibility of the condition in elderly females who have vague abdominal pains associated with intermittent pain in the thigh muscles.

Remarkably, some patients may suffer for as many as twenty-six years from repeated attacks of colic, due no doubt to semi-strangulation, without ever going on to complete strangulation, as was the case reported by Zwalenburg in 1913, of a woman of 76, who was operated upon for a double obturator hernia.

Curiously enough, the diagnosis of a strangulated femoral hernia has been made in cases where an enlarged tender gland occupied the crural canal. Such

a case was reported by Anderson in 1900. A married woman aged 75 was operated upon for what was supposed to be a strangulated femoral hernia; the crural canal was explored, and the small lump which had been felt proved to be an enlarged gland. The wound was closed, and later on the same day the obturator region was explored, a hernia found and reduced, and the sac excised, the patient making an uninterrupted recovery.

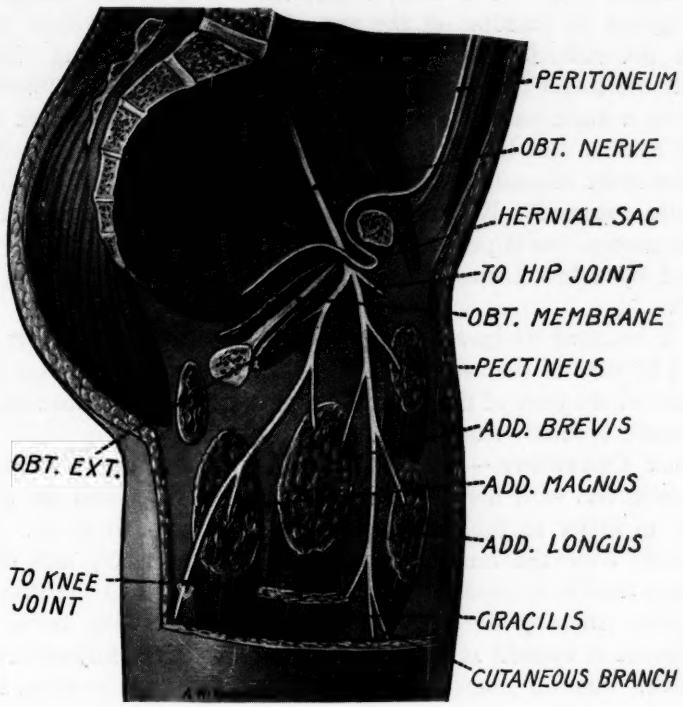

FIG. 364.—Diagrammatic drawing showing the relation of an obturator hernial sac to the obturator nerve. The distribution of the nerve to the thigh muscles is also seen.

The contents of the sac are usually small intestine, but cases are recorded in which the bladder and Fallopian tube have been found in the hernial sac. Gladstone gives a very complete description of such a case, which was found in a dissecting-room subject 78 years old.

TREATMENT

There is only one form of treatment for this type of hernia, and that is by operation. The application of trusses must be condemned even in the simple variety of hernia.

Taxis is more dangerous in obturator hernia than in any other form of hernia, as in cases of strangulation gangrene may set in quite early and there is therefore a grave risk of rupturing the gut.

As far as the operative procedure is concerned there are two methods of approach, the abdominal and the obturator, and in some cases a combined operation is required.

Abdominal Operation.—This is the method of choice, as thereby the surgeon is enabled to deal with any complication which may arise. It is the route which has been used in the majority of cases in the past, as in quite a number of cases the diagnosis was not made until a laparotomy had been performed. A subumbilical paramedian incision is used, and the abdomen opened by retracting the rectus muscle laterally. The Trendelenburg position is essential, as in this position all the possible hernial sites can be easily inspected. An obturator hernia when found can be investigated by packing off the rest of the intestines with a hot towel. If the hernia is not reducible by gentle traction, the constricting ring should be stretched with the finger; and if this does not suffice, then the fibrous ring must be divided with a knife passed along a grooved director, taking care not to divide the obturator vessels and nerve. If this does not allow retraction of the bowel, then an incision must be made in the obturator region and the hernia carefully helped back by gentle pressure. When the gut is reduced it must be inspected; if gangrene of a portion has supervened, then, if it is small, the portion of bowel can be invaginated by a few Lembert sutures, while if of larger area, resection must be performed.

The sac is inverted by inserting a pair of pressure forceps into it and grasping its fundus. The base of the sac is transfixed with catgut and the sac itself excised. The cut surface of the base of the sac is then sutured to the peritoneum surrounding it. The abdomen is finally closed in layers.

Obturator Operation.—When operation by the obturator route is undertaken, the patient lies with the pelvis raised on a sand-bag, and the thigh is flexed and abducted in order to relax the pull of the adductor muscles. An adequate incision is made over the hernial protrusion and the fascia lata divided. The adductor longus muscle is exposed and retracted medially and the pectineus muscle retracted laterally, thereby allowing the sac to come into view between these two muscles. The sac is opened at its fundus, and its contents inspected and returned to the abdomen. Should gangrene have occurred, laparotomy must be performed at once and the gangrenous gut resected. The sac should be cleaned by dissection, transfixed at its neck, and excised. It is often advisable to take a portion or the whole of the pectineus muscle and stitch it to the periosteum surrounding the obturator canal. This effectually blocks the canal and helps to prevent a recurrence. The wound is then closed without drainage.

The Inguinal Route for the Radical Cure of Obturator Hernia.—During the past twenty years there has been a tendency to operate on femoral herniæ by an inguinal approach, and Milligan has advocated an inguinal route for obturator hernia. Such a route should not be attempted if there is any fear of strangulation.

An oblique incision is made parallel to, and just above the inner end of, Poupart's ligament, thereby exposing the external abdominal ring and the attachment of the external oblique aponeurosis to Poupart's ligament. An incision is then made in the aponeurosis and its upper part retracted. The exposed spermatic cord is drawn upwards so as to expose the external iliac vein and the femoral ring. The peritoneum is carefully pushed upwards and held out of the way by retractors, whereupon the obturator vessels and nerve can be seen entering the obturator foramen and any hernial sac can be observed. The sac of the hernia is drawn from the foramen and opened, and its contents, if any, returned to the abdomen; the neck of the sac is then transfixed and the sac excised.

By this method the possibility of adhesions forming in the region of the obturator foramen is avoided, but the exposure is not nearly so effectual as by the abdominal route.

REFERENCES

ANDERSON, G. R., *Liverpool Med.-Chir. Jour.*, 1900, xx, 271.
COULSON, W., *Lancet*, 1863, ii, 303.
GLADSTONE, R. J., *Ann. of Surg.*, 1901, xxxiv, 796.
GODLEE, R. J., *Lancet*, 1885, i, 655.
HILTON, J., *Med. Chir. Trans. Lond.*, 1848, xxxi, 323.
KING, W., *Lond. Med. Gaz.*, 1842, xxxi, 409.
MILLIGAN, E. T. C., *Brit. Med. Jour.*, 1919, ii, 134.
PICQUÉ, L., and POIRIER, P., *Rev. de Chir.*, 1891, xi, 956.
PIMBET, D., *Thèse de Paris*, 1882.
SINIGAR, H., *Brit. Med. Jour.*, 1897, i, 587.
WATSON, L. F., *Hernia*, 2nd ed., 1938. London: Henry Kimpton.
VAN ZWALENBURG, C., *Surg. Gynecol. and Obst.*, 1913, xvi, 422.

SIMPLE NON-SPECIFIC ULCER OF THE CAECUM

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Cases of ulcer of the cæcum which are non-specific and simple in nature are relatively rarely encountered.[71] They are of uncertain aetiology,[67] their symptoms are variable, and even when they have perforated, they have never been diagnosed with certainty previous to operation. They give rise to severe or fatal complications if untreated.

For these reasons a short account of the condition and the report of a personal case appears to be worthy of record.

CASE REPORT

History.—James A., a quarrier aged 32, was admitted to the Royal Infirmary, Edinburgh, on April 27, 1936, as an urgent surgical case, with the diagnosis of acute appendicitis.

He complained of severe abdominal pain of twelve hours' duration and of nausea for eight hours, and was retching on admission, but there was no actual vomiting. His symptoms were increasing in severity.

His past history revealed no illnesses of any kind, and there was nothing of note in his family history—such as ulcer diathesis, tuberculosis, cancer, syphilis, or nephritis. He was well the previous day, and his illness started at 9.15 a.m. on the day of admission, with pain commencing and remaining in the midline just below the umbilicus. This was followed by increasingly severe nausea and retching.

He had never been troubled by constipation and did not use aperients, and had not before experienced either abdominal pain or nausea. He had not studied his stools, but they had never occasioned him any discomfort by undue hardness. There were no urinary symptoms. His diet in quality and quantity was in no way unusual.

On Examination.—The patient was slim but well developed, and tended to retch occasionally. There was marked tenderness in the right iliac fossa, localized at McBurney's point, and rigidity of the muscles of the right lower quadrant of the abdomen. There was no hyperæsthesia. The pulse was 80 and the temperature 99·2° F. The urine was normal. Rectal examination before admission elicited slight tenderness, which was not localized; subsequent to admission there was no tenderness.

On dental examination there was a marginal gingivitis, especially of the lower gums in the incisor region. The teeth themselves were in good condition, but there was some absorption of the bone around the lower incisors. There was no gross pyorrhœa.

A pre-operative diagnosis was made of acute appendicitis.

At Operation.—At 10 p.m. the same evening, under general anæsthesia, the abdomen was opened through a gridiron incision in the right iliac fossa by the method advocated by George L. Chiene,[46] which gives a wider exposure than the ordinary gridiron. The external oblique was divided in the line of its fibres, and the anterior and posterior sheaths of the rectus muscle were then incised vertically, the nerves being seen and preserved. This gave a free exposure of the cæcum, which was delivered at the wound. The appendix was healthy. There was an ulcer on the anterior wall of the cæcum on a level with the ileocæcal valve and about 2 cm. distant from it. It felt thick and hard and had a crater on the mucous surface which admitted the tip of the index finger. The peritoneum over it was slightly reddened. There was no perforation and no peritonitis. An occlusion clamp was applied to the cæcum and the ulcer excised; the cæcum was then closed in two layers with catgut. The appendix was removed in the usual manner and the stump invaginated.

No enlarged glands were noted; the ascending colon and terminal ileum were normal on inspection and palpation. The pylorus and pelvic colon were normal on palpation. The abdomen was closed in layers without drainage.

SUBSEQUENT PROGRESS.—The wound healed normally, and on the tenth day the stitches were removed and the patient was allowed up; he was discharged on the thirteenth day. The temperature was normal throughout and the bowels moved daily, except on the second day. The fæces were normal in all respects. Urea-nitrogen estimation by the urease method was 14 mg. per cent. The report of smears from the ulcer taken at operation was: "No tubercle bacilli were found, organisms were very scanty, only one or two intestinal bacteria being found".

FIG. 365.—Drawing of solitary, simple ulcer of the cæcum from the author's case.

DESCRIPTION OF SPECIMEN.—The specimen consisted of a portion of the anterior cæcal wall, lateral to the anterior longitudinal band at the level of the ileocæcal junction. It measured 2·5 × 2 cm. in diameter and 0·5 cm. in thickness. On the mucous surface there was an ulcer 2 cm. in diameter having a crater 1 cm. in diameter and a thickened rolled edge. The base of the ulcer was of a greenish colour and necrotic, and the walls were a deep bluish-red (*Fig.* 365). There was slight reddening of the peritoneum over the ulcer. There was no gross evidence of peritonitis.

FIG. 366.—Low-power microphotograph of section of ulcer of the cæcum. (× 5.)

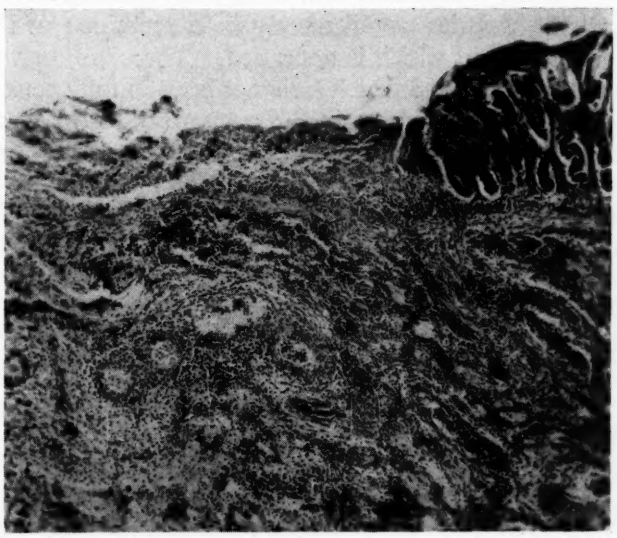

FIG. 367.—High-power microphotohraph of section of ulcer of the cæcum. (× 90.)

On *histological examination* the ulcer was a simple localized ulcer showing no sign of malignancy, tuberculosis, or syphilis (*Fig.* 366). Its base was formed of inflammatory tissue rich in polymorphs. The vessels were all markedly dilated, the walls showed no endarteritis, and no thrombosis was noted. The ulceration extended through the mucous membrane and submucosa, but the muscle was intact. The base of the ulcer and the walls were œdematous. In the submucosa and muscular layers there was a considerable amount of fibrin present and areas of hæmorrhage, but little evidence of fibrous connective-tissue production (*Fig.* 367). The serosal surface was healthy apart from moderate vascular dilatation in the subserosa.

DISCUSSION

Simple ulcer of the large bowel was recognized as an entity as far back as 1830, when Cruveilhier first described the condition in his *Atlas d'Anatomie et Pathologie*.[1]

There is now over a century of literature on the subject, in which 68 cases have been recorded, and the subject has been reviewed by, amongst others, Quénu and Duval in 1902,[27] Soupalt in 1920,[34] Barron in 1928,[54] Wise in 1931,[60] and Wilkie in 1937.[69]

The ulcers have been described in text-books and journals as the same disease whether occurring in the cæcum, colon, or rectum, and there is a tendency to link them up with ulcers elsewhere in the gastro-intestinal tract, both in their aetiology and behaviour.[54]

Of these ulcers the majority (42 cases) have occurred in the cæcum or ascending colon, the most frequent site being the cæcum (23 cases), and it is with the cæcal ulcers that this communication is mainly concerned and of which the author's case is an example (*see* table on p. 529).

Aetiology and Pathology.—The theories of the causation of such ulcers of the large bowel are similar to, and as varied as, those of ulcers elsewhere in the gastro-intestinal tract. They include inflammatory, circulatory,[13, 49] neurogenic, bacteriological,[47, 68] toxic,[25] digestive, and purely mechanical explanations.[26, 34, 47]

To discuss all the theories would merely be a repetition of the literature on the subject, a bibliography of which is appended. There are, however, two points which are sufficiently striking and which have not been so fully considered—namely, the situation of the majority of ulcers in the cæcum (23 cases), and their situation in the cæcum on a level approximately with the ileocæcal valve. In this they bear an anatomical as well as a pathological relationship to ulcers of the duodenum. In both the ulcer develops opposite to a segment of gut guarded by a valve or sphincter, having a similar and connected innervation; in both the blood-supply of the affected area is relatively deficient, particularly when the bowel is distended,[54] the vasa brevia and longa[66] (anterior and posterior cæcal arteries) terminating or inosculating with little anastomosis, as does the artery supplying the "anæmic spot" on the anterior duodenal wall.[30] The slightly acid *p*H reaction of the fæces[53, 56] in the terminal ileum, which delivers its contents towards this segment, and the high incidence in males, further strengthens the analogy.

Hence, whatever may be the primary factor—thrombosis,[26] embolism,[68] local endarteritis,[27] fissure formation,[52] lymphoid degeneration, or actual pressure necrosis from hardened and roughened fæces[50]—it is easy to appreciate why such a large number occur at this site, fail to heal, and frequently perforate. The regional distribution of the disease is not of particular significance to-day, though formerly inflammatory bowel conditions were commoner in grain-eating people, and it is of

interest that a large proportion of the recorded ulcers have occurred in Scotland[69] and France. " Coprolithes d'Écosse où l'on mange beaucoup d'avoine "[51] is apparently a teaching axiom of Barcelona and certain Continental schools.

RÉSUMÉ OF REPORTED CASES OF CÆCAL ULCERS

No.	Author	Sex	Age	Diagnosis	Perforation	Operation	Result
1	Bradbury[15]	M.	30	None	—	None	Died. P.M.
2	Parker[17]	F.	49	None	Yes	None	Died. P.M.
3	Ollier[18]	M.	56	Peritonitis	Yes	None	Died. P.M.
4	Morel and Scheyron[19]	M.	46	Cæcal cancer	—	Extensive resection. Hemi-colectomy	Cured
5	Southam[20]	M.	67	None	Yes	None	Died. P.M.
6	Tedenat[28]	M.	52	None	Yes	Drainage	Died. Incomplete P.M.
7	Bazy[35]	M.	20	Appendicitis	Yes	Closure	Cured
8	Sieur[37]	M.	30	None	Yes	None	Died
9	Lardennois[38]	M.	52	Peritonitis	Yes	Closure	Died. P.M.
10	Levy[39]	F.	Girl	Appendicitis	—	Invaginated	Cured
11		M.	—	Appendicitis	Yes	Peritonization	Died. P.M.
12	Dickinson[41] (3 cases)	M.	22	Chronic appendicitis or cholecystitis	Yes	Laparotomy. Cholecystostomy	Died. P.M.
13		M.	56	Appendicitis	Yes	Cæcum sutured to abdominal wall and drainage	Cured
14	Bowen[42]	F.	27	None	Yes	Suture failed	Died. None
15	Mintz[44]	F.	18	Intussusception	—	Resection	Cured
16	Moll[48]	M.	21	Acute appendicitis	—	Local resection	Cured
17	Barron[54]	F.	23	Acute appendicitis	—	Oversutured	Cured
18	Bombi[55]	M.	37	? Cæcal ulcer	—	Local resection	Cured
19	Thaler[63]	F.	38	Acute appendicitis	Yes	Drainage	Died. P.M.
20	Petridis[67]	F.	33	Acute dilatation of stomach	Yes	None	Died. P.M.
21	Author's case	M.	32	Acute appendicitis	—	Local resection	Cured

Mortality with non-operative treatment 100 per cent
Mortality with operative treatment 40 „ „
} Total mortality, 57 per cent

Note.—In addition to the ulcers in the above table, Osler states " two instances of ulcer of the cæcum, both with perforation, have come under my observation ". There does not appear to be any other record containing fuller details of these in the literature.

This makes a total of 23 recorded cæcal ulcers. Frequency of perforation, 65 per cent.

In the cases recorded the situation of all cæcal ulcers is not exactly stated, but in all except one the ulcer is situated either opposite the valve or on the anterior wall on a level with it.

The histology of the condition in some instances showed vascular lesions around the ulcer, endarteritis,[43] and thrombosis[13, 26]; according to the stage of ulceration, absence of epithelium, submucosa, and even muscle may be the feature. There may be a thickening of the serosa, with newly formed and dilated vessels.[44] Round-celled infiltration, with polymorphs and phagocytes; atrophy of muscle; and in some cases increase of connective tissue, indicate varying stages of inflammation, destruction, and repair.

Clinical Features.—There are no pathognomonic signs to aid in the diagnosis of cæcal ulcers. The symptoms are usually those of appendicitis; the pain,

however, is fixed and constant in one situation. The exact site is usually at or near McBurney's point, and in the case recorded was very slightly nearer the umbilicus. Constipation is present in many of, but not all, the cases, and was not a feature of the author's case; moreover, the incidence of cæcal ulcers is twice as common in males as in females. Melæna is occasionally present. The age incidence is not of particular significance, varying between 18 and 67 years, the average age being 37 years.

The complications are those of perforation and peritonitis, which may result in a general peritoneal infection or a localized abscess, as occurred in over half (15) of the cases.

The provisional or differential diagnosis in the cases reviewed has been as follows: Appendicitis, 8 cases; peritonitis, 3 cases; appendicitis or cholecystitis, 1 case; cæcal carcinoma, 1 case; intussusception, 1 case; acute dilatation of the stomach, 1 case. In 6 cases there was no diagnosis, and in 1 case a radiological diagnosis of cæcal ulcer, with a filling defect of the cæcum, was made.[55]

Treatment.—There is no record of any case recovering without operation. Of the total 21 cases (excluding Osler's 2 cases, where details are not available), 9 out of the 15 cases submitted to operation recovered and 6 died, and the remaining 6 unoperated upon all died. The operative procedure adopted depended on the state of the ulcer, its size, and the presence or absence of peritonitis, and whether there was doubt as to its simple nature.

The methods performed were: in perforated cases—simple suture, using linen, silk, or catgut; in non-perforated cases—peritonization by Lembert sutures, or local resection of the ulcerated part of the cæcum or extensive resection of the right half of the large intestine from ileum to transverse colon.

It would seem, however, in view of the capacious nature of the cæcum, that localized resection of the ulcer should be practicable and sufficient in many cases, and where perforation and peritonitis have occurred drainage in addition would be necessary.

CONCLUSION

A typical case of simple non-specific solitary ulcer of the cæcum treated by excision of the ulcer, with cure, is recorded. The aetiology, clinical features, and treatment are briefly reviewed, and it is shown that ulcer of the cæcum has many features in common with duodenal ulcer.

I wish to express my thanks to Mr. George L. Chiene for permission to deal with and record the case; to Miss Thom for the drawing; and to Mr. D. Smith and Mr. D. Grant for the sections and microphotographs.

BIBLIOGRAPHY

[1] CRUVEILHIER, J., *Atlas d'Anatomie et Pathologie*—Rectum, 1830–32, 4.
[2] MARCHESSAUX, *Bull. Soc. Anat. de Paris*, 1837, xii, 343.
[3] ROGEE, *Ibid.*, 1838, xiii, 181.
[4] GREENHOW, *Pathol. Soc. Newcastle and Gateshead*, 1848, December.
[5] CLAUS, *Inaug. Diss.*, Zurich, 1856.
[6] LEBERT, *Traité d'Anatomie et Pathologie générale et spécielle*, 1861, ii, 206.
[7] DUJARDINE, *Soc. de Sci. méd. et naturelle de Bruxelles*, 1864.
[8] DICKINSON, *Trans. Pathol. Soc. Lond.*, 1867, xviii, 101.

[9] ROBIN, *Thèse de Paris*, 1869.
[10] COSTIN, *Brit. Med. Jour.*, 1870, ii, 247.
[11] LEPINE, *Bull. Soc. Anat. de Paris*, 1870, xlv, 27.
[12] CHOUPE, *Ibid.*, 322.
[13] KOCHER, *Deut. Zeits. f. Chir.*, 1877, viii, 331.
[14] MICKLE, *Brit. Med. Jour.*, 1883, i, 637.
[15] BRADBURY, *Lancet*, 1884, ii, 637.
[16] OLIVER, *Ibid.*, 1885, i, 424.
[17] PARKER, *St. Bart.'s Hosp. Rep.*, 1887, xxiii, 213.
[18] OLLIER, *Loire méd.*, 1892, xi, 3.
[19] MOREL and SCHEYRON, *Soc. de Méd. de Toulouse*, 1895.
[20] SOUTHAM, *Brit. Med. Jour.*, 1895, i, 254.
[21] GUINARD, *Thèse de Paris*, 1896, No. 324.
[22] MOREL and RISPAIL, *Soc. de Méd. de Toulouse*, 1896.
[23] DEMANGE, *Rev. méd. de l'Est*, 1897, Aug., 500.
[24] GAMGEE, *Brit. Med. Jour.*, 1898, i, 950.
[25] GANDY, *Thèse de Paris*, 1899.
[26] PRUTZ, *Arch. f. klin. Chir.*, 1900, lx, 323.
[27] QUÉNU and DUVAL, *Rev. de Chir.*, 1902, xxvi, 692.
[28] TEDENAT, quoted by Quenu and Duval, *Rev. de Chir.*, 1902, xxvi, 825.
[29] ZICKLER, *Beitr. z. klin. Chir.*, 1909, lxvii, 189.
[30] WILKIE, D. P. D., *Surg. Gynecol. and Obst.*, 1911, 399.
[31] LYLE, *Ann. of Surg.*, 1912, lvi, 473.
[32] OSLER, Sir WM., *Principles and Practice of Medicine*, 1915, 521. New York and London: Appleton & Co.
[33] EUNICKE, *Deut. Zeits. f. Chir.*, 1918, cxlvii, 284.
[34] SOUPALT, *Rev. de Chir.*, 1920, lviii, 480.
[35] BAZY, *Bull. et Mém. Soc. de Chir. de Paris*, 1920, xlvi, 416.
[36] GREGOIRE, *Ibid.*, 420.
[37] SIEUR, *Ibid.*, 421.
[38] LARDENNOIS, *Ibid.*
[39] LEVY, *Deut. Zeits. f. Chir.*, 1921, clxv, 366.
[40] ECOT and RICHARD, *Bull. et Mém. Soc. de Chir. de Paris*, 1922, xcii, 138.
[41] DICKINSON, *Jour. Amer. Med. Assoc.*, 1922, lxxviii, 1792.
[42] BOWEN, *Guy's Hosp. Rep.*, 1922, lxxii, 441.
[43] CHALIER and MALLET-GUY, *Arch. de Mal. de l'App. digestif*, 1923, xiii, 521.
[44] MINTZ, *Zentralb. f. Chir.*, 1923, l, 1208.
[45] BROERE, *Nederl. Tijds. v. Geneesk.*, 1923, lxvii, 2274.
[46] CHIENE, G. L., *Handbook of Surgery*, 1923, 487. Edinburgh: Livingstone.
[47] DELAFIELD and PRUDDEN, *Textbook of Pathology*, 12th ed., 1923, 775. Baltimore: Wood & Co.
[48] MOLL, *Zentralb. f. Chir.*, 1926, liii, 2274.
[49] BOSS, *Beitr. z. klin. Chir.*, 1927, cxl, 401.
[50] OERTEL, *Outlines of Pathology*, 1927, 77. Montreal: Renouf.
[51] ROSELL and CAMBIES, *Coprologie clinique*, 1927, 45. Paris: Vigot Frères.
[52] RANKIN, *Surgery of Colon*, 1926, 104. New York: Appleton & Co.
[53] REDMAN, T., WILLIMOT, P. G., and WOKES, F., *Biochem. Jour.*, 1927, xxi, 589.
[54] BARRON, *Arch. of Surg.*, 1928, xvii, 355.
[55] BOMBI, G., *Policlinico* (Sez. Prat.), 1929, xxxvi, 1550.
[56] REDMAN, T., *Biochem. Jour.*, 1929, xxiii, 256.
[57] BOYER and TUFT, *Jour. Amer. Med. Assoc.*, 1930, May, 1566.
[58] BARRON, *Surg. Gynecol. and Obst.*, 1930, l, 870.
[59] SAKORRAPHOS and PHOTAKIS, *Deut. Zeits. f. d. g. gericht. Med.*, 1930, xv, 455.
[60] WISE, *Ann. of Surg.*, 1931, xciii, 1273.
[61] LIND, *Ohio State Med. Jour.*, 1931, xxvii, 621.
[62] WISE, *Bull. Med. Univ. Maryland*, 1934, xxiv, 45.
[63] THALER, *Münch med. Woch.*, 1935, lxxxii, 255.
[64] KUMMER, *Nederl. Tidjs. v. Geneesk.*, 1935, lxxix, 2621.
[65] DELANNOY, *Echo méd. du Nord.*, 1935, iii, 901.
[66] BEESLY and JOHNSTON, *Manual of Surgical Anatomy* (rev. Bruce and Walmsley), 1935, 422. London: Oxford University Press.
[67] PETRIDIS, *Bull. Soc. d'Obst. et de Gynecol.*, 1936, xxv, 505.
[68] MAYO, C. H., ROSENOW, *Jour. Amer. Med. Assoc.*, 1922, lxxviii, 1793.
[69] WILKIE, Sir D., *Surgery*, 1937, i, 655.
[70] CORACHAN, M., *Chirurgie del Colon.* Barcelona.
[71] ROMANIS and MITCHINER, *Science and Practice of Surgery*, 5th ed., 1937, ii, 653.

CARCINOMA OF THE JEJUNUM: ACUTE INTESTINAL OBSTRUCTION

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THE rarity of carcinoma of the jejunum cannot but have impressed itself on both the physician and the pathologist. The figures from the Mayo Clinic accentuate its infrequent occurrence: there, between 1921 and 1930, although 2513 cases of carcinoma of the stomach, and 2767 cases of carcinoma of the rectum were seen, no more than 8 cases of jejunal cancer came under observation; and at the Vienna General Hospital the figures are even more striking, for in a series of 41,838 autopsies, although 8535 cases showed carcinoma, yet in only 8 cases was the small intestine the site of the growth.

Clearly certain factors, such as the alkaline reaction, the fluid nature, and the lack of stasis of the contents of the area, are responsible for preventing the spread of neoplastic tissue in the intestinal tract; and the behaviour of pyloric carcinoma, which freely infiltrates the stomach wall, but almost invariably declines to affect the duodenum, supports the possibility that these factors may be causative in the rarity of jejunal cancer.

Malignant disease affecting the jejunum is of various types: lymphosarcomatosis, localized lymphosarcoma, leiomyosarcoma, myosarcoma, secondary carcinoma (from a primary focus in the ovary, uterus, or stomach)—all of them relatively more common than primary carcinoma, which latter may be differentiated as adenocarcinoma conforming to Broder's Class I or alternatively as of a comparatively undifferentiated type. The tumour tends to be polypoid in the younger patient, with resulting acute obstruction from intussusception. The usual form, however, is the slowly growing ring neoplasm of the scirrhous type, and this normally either ulcerates or undergoes colloid degeneration.

The condition frequently described as carcinoid tumour of the small intestine was formerly regarded as a basal-celled carcinoma (Johnson), but is now considered to be an argentaffin tumour, arising from the cells of Kulchitsky (Masson), or possibly a neurocytoma from the cells of Auerbach's plexus (Ehrlich).

The pathology of carcinoma of the jejunum could not be more tersely put than in the words of d'Allaines: "En général, l'épithélioma du grêle donne assez bien à la palpation la sensation que donne un petit 'bouton de Murphy' placé au niveau d'une anastomose". "L'extension de l'épithélioma est assez lente et pendant longtemps il reste confiné aux territoires ganglionaires immédiatement adjacent." And a recent communication from Dukes on carcinoma of the rectum, which emphasizes the lengthy interval before secondary glandular metastasis occurs in that disease, strikes a pleasing note of similarity with the words above.

CLINICAL ASPECT

About 200 cases in all have been recorded in the literature. The average age of the series of cases collected by Mayo and Nettrour, and in d'Allaines' series of

114 cases, was between 30 and 40, the youngest being 28. Staemmler has reported 4 cases of malignant disease of the small intestine in persons under 20, and Badile has reported 1 case in a boy of 13. Hoffman's case is of a man of 28 with a polypoid neoplasm. Duncan's case of malignant disease of the small intestine in a boy of 3½ years is the youngest ever recorded.

The principal complications of the condition are chronic and acute intestinal obstruction and perforation: the latter is extremely rare. Venot and Parcellier were able to collect only two instances of perforation, the first into the peritoneal cavity, and the second with a concomitant jejuno-colic fistula, and Woelcher's case case had a colic fistula complicating.

DIAGNOSIS

The fluid content of the jejunum results necessarily in the extraordinary rarity of acute obstruction as a manifestation of the disease; and a case of this type in a man of 27 seemed to me adequate to merit record. This condition is not one which can be diagnosed with certainty before operation; but if a patient of carcinoma age presents clinically a picture of high intestinal obstruction, with negative radiological evidence of disease of the stomach or duodenum, a progressive anæmia, occult blood in the stools, and a persistent loss of weight, then laparotomy is warranted, for malignant disease is a very real possibility. The surgeon must of course bear in mind those other causes of high intestinal obstruction, such as bands, kinking, duodenal ileus, tuberculous stricture, chronic regional enteritis, and lymphosarcoma.

The obstructive symptoms are present for a period of time which varies up to six months, and may occur commonly some three or four hours after meals. Abdominal cramps are referred to the subumbilical region, and are associated with a feeling of distension which is relieved by vomiting (Rivers considers that the subumbilical region is the site for referred pain from jejunal regions). While the vomiting is of course severe in the case which presents as an acute obstruction, it is more characteristic if it be slowly progressive in intensity. Should the vomit contain bile, it is increasingly likely that the responsible lesion is below the second part of the duodenum. Constipation is variable, as would be expected from the level of the obstruction, and is therefore not of great diagnostic value. Occult blood in the fæces, however, is an almost invariable finding, and according to Mayo is frequently the first definite evidence of organic disease. Anæmia is a marked feature, and the hæmoglobin may descend so low as 35 per cent, with a red-cell count of 3,000,000. Plunkett and Foley suggest that the macrocytic element in the blood-picture is the result of the failure of the diseased intestinal wall to absorb enough of the hæmopoietic substance postulated by Castle; and the microcytic element from the failure of iron absorption in the general state of malnutrition. Numerous cases of carcinoma of the small intestine, cæcum, and ascending colon are consequently considered clinically to be pernicious anæmia before the correct diagnosis is ultimately established.

PHYSICAL SIGNS

Visible peristalsis was noted in all the 3 cases recorded by Johnson, and he emphasizes the significance of this finding. The characteristic radiographic distension and transverse striations of small-intestine obstruction was seen in one of the

cases here described, but it is very exceptional to obtain such clear evidence without a barium-meal examination. A positive radiological diagnosis was made in 10 per cent of Mayo's cases, the appearance being usually a stenosis of the intestinal lumen at the site of the carcinoma, with a proximal dilatation.

CASE REPORTS

Two cases of carcinoma of the jejunum admitted under the care of Mr. J. B. Ferguson Wilson and a third case under the care of Prof. Graham Simpson, showed unusually interesting features, and we felt were worthy of detailed record. The first was associated with acute obstruction and occurred in a remarkably young patient; the second was typical of the disease, and the X-ray appearances before operation were highly instructive; the third case occurred also in a young patient.

Case 1.—

HISTORY.—The patient, a young man 27 years of age, was admitted as an "acute abdomen". He had a history of 20 hours' severe colicky pain, periumbilical and hypogastric in situation. Vomiting occurred three hours after the onset of the pain, and at frequent intervals afterwards. The previous history extended over six weeks, and was one of epigastric pain occurring one hour after food, but not influenced by the type of food taken nor associated with vomiting. There was increasing constipation for the same length of time.

ON EXAMINATION.—There was marked rigidity and local tenderness in the hypogastrium, but there was no distension, no palpable mass, and no visible peristalsis. The patient looked ill, and clearly the pain was severe. A tentative diagnosis of high intestinal obstruction with an associated internal strangulation was made.

AT OPERATION.—I opened the abdomen under spinal anæsthesia (2 c.c. heavy percaine), using a central right paramedian incision, and found a rounded mass of about 4 in. in diameter lying in the hypogastrium. This proved to be a segment of jejunum containing a neoplasm of the ring type. One area of the mass was deep purple in colour, and close inspection revealed that the centre of this area was necrotic, with absence of peritoneum over it, and with the underlying dark red soft mass of tumour showing through. This recent peritoneal involvement was no doubt the cause of the marked tenderness and hyperæsthesia found clinically. The affected segment was excised, and a side-to-side anastomosis was performed, the related glands being too extensively involved to permit removal.

SUBSEQUENT PROGRESS.—Two days later the patient's pulse-rate rose and he began to vomit copious amounts of fluid. A diagnosis of post-operative paralytic ileus was made. Continuous duodenal aspiration, using a nasal tube and the author's apparatus, together with continuous intravenous drip saline, was instituted. Within three days the patient's condition improved, and he subsequently made an uninterrupted recovery. At the moment of writing he is undergoing a course of deep X-ray therapy to the abdominal glands.

PATHOLOGICAL REPORT (by kind permission of Howard J. Barrie, Pathologist, The Royal Hospital, Sheffield).—

Macroscopical (*Figs.* 368, 369).—An opened piece of jejunum 29 cm. long and 8 cm. in circumference, with a narrow fringe of mesentery attached. The proximal end is identifiable by the fact that its terminal 4 cm. has been left unopened by the surgeon. At a distance of 14 cm. from this end the mucosa is raised over a roughly circular underlying disc of growth 4 cm. in diameter, on the summit of which there is a shallow ulcer 3 by 2·5 cm. with sloping edges and a purplish, necrotic concave base. At a point 4·5 cm. distal to this growth one valvulus shows a slight fusiform localized thickening, and but 4 cm. from the distal end there is a submucosal nodule of growth 0·6 cm. in diameter just erupting through the mucosa, with no visible connections with the muscularis. The muscular wall is 0·25 cm. above and below the growth.

The serosa is very injected, becoming black towards the growth, and opposite the base of the ulcer it exhibits a stellate perforation, with gangrenous edges. Opposite the ulcer there are several slightly enlarged glands in the attached mesentery, with obvious involvement by growth. There is a chain of very firm slightly enlarged glands along the rest of the mesentery, some with grey cellular, some with firm pale white, surface.

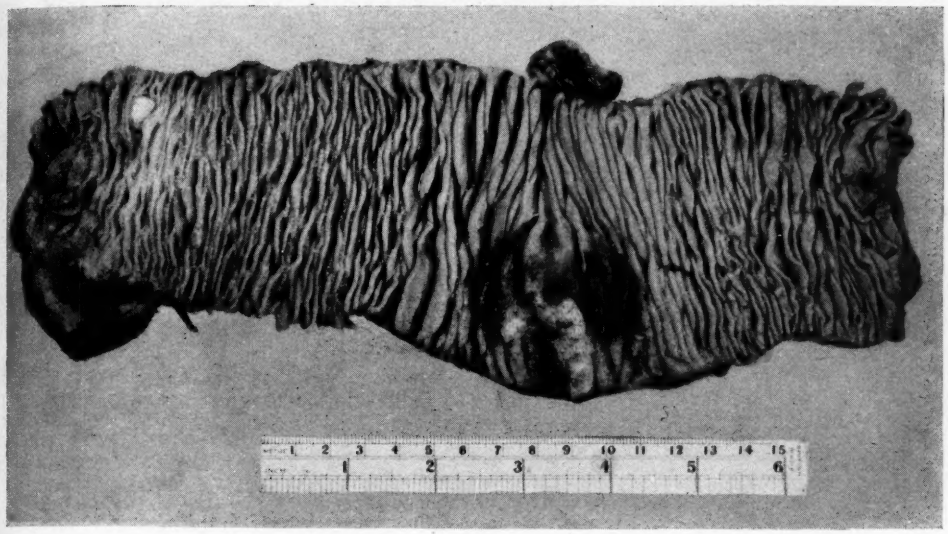


FIG. 368.—*Case* 1. The mucous surface of the jejunum affected by the neoplasm.

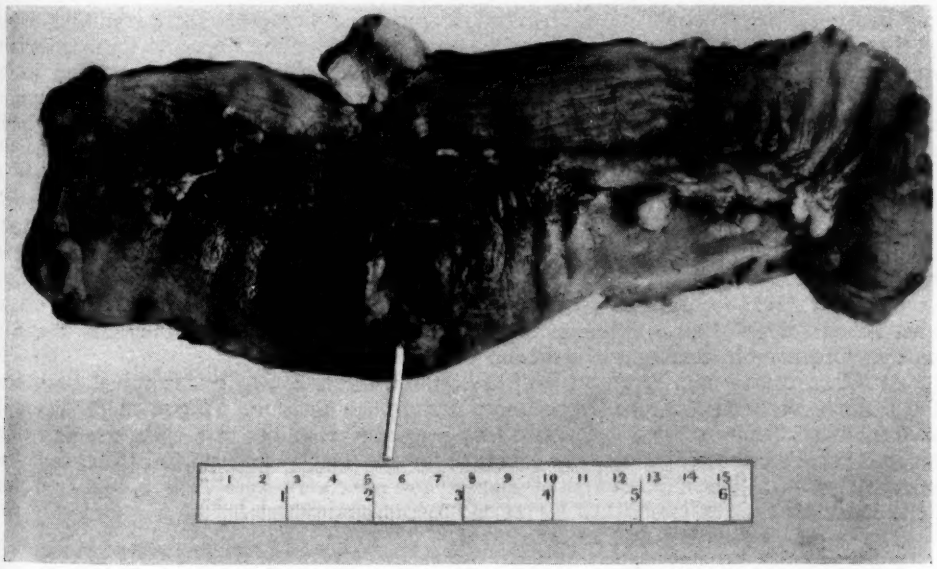
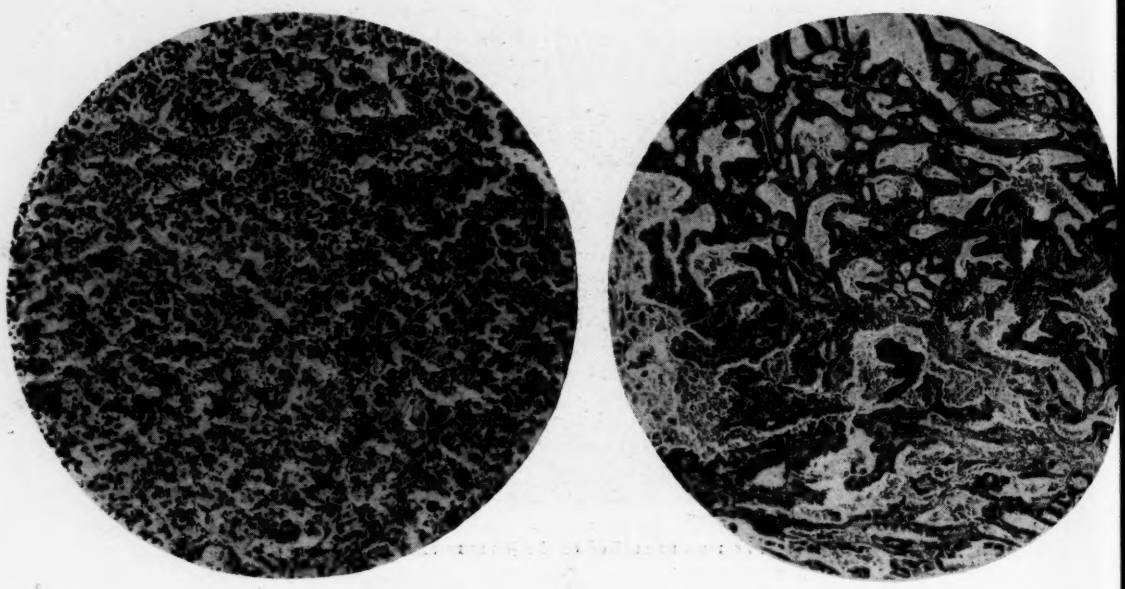


FIG. 369.—*Case* 1. Outer aspect of the affected segment of jejunum, showing the site of incipient perforation.

Microscopical (*Figs.* 370, 371).—The main growth and the two outlying nodules all show spheroidal-celled carcinoma. The mesenteric glands opposite the outlying nodules are chronically inflamed, but contain no carcinoma.



FIGS. 370, 371.—*Case* 1. Microscopical appearance. Low and high power.

Case 2.—

HISTORY.—The patient was a woman 53 years old, first admitted under the care of Dr. C. Gray Imrie, in October, 1937. She complained that for two years she had suffered from epigastric pain occurring some four hours after meals. The pain was accompanied by "a lump in the stomach" and "gurgling sounds inside"; it was relieved by vomiting, which was frequently copious in quantity. She dared not eat solids of any kind, as these invariably precipitated a severe attack. Anorexia appeared, together with a choking sensation in her throat on exercise or swallowing of fluids, and the bowels, formerly regular, were becoming increasingly constipated. During this time she became pale in appearance, and the body-weight diminished by 3 st.

ON EXAMINATION.—Clinically the abdomen showed no abnormality, there being no visible peristalsis, no palpable tumour, and no local tenderness. Occult blood was repeatedly present in the fæces, and the blood-picture showed a hæmoglobin percentage of 66, and a red-cell count of 3,880,000. X-ray examination following a barium meal revealed no abnormality of the stomach, duodenum, or colon, but at a subsequent investigation there was present a single, large, distended loop of small intestine, with the classical distension and transverse striations of small-intestine obstruction (*Fig.* 372). The pre-operative diagnosis was therefore chronic small-gut obstruction of unknown cause.

AT OPERATION.—Mr. Ferguson Wilson explored the abdomen under spinal anæsthesia (2 c.c. heavy percaine), using a right upper paramedian incision. About 12 in. from the duodenojejunal flexure there was found a ring neoplasm, resulting in a mass the size of an orange. Some mesenteric glands were enlarged, but the liver was considered to be free from secondary growth. The affected area of jejunum and mesentery were excised, and continuity of the intestinal tract re-established by an end-to-end anastomosis.

The patient's recovery was uninterrupted.

PATHOLOGICAL REPORT.—

Macroscopical.—An opened piece of jejunum 16 cm. long, 7 cm. circumference, with a narrow fringe of mesentery attached. Situated centrally in regard to the long axis there is a firm ring of growth well demarcated from the surrounding mucosa, projecting 1 cm. into the lumen, and extending transversely for three-quarters of the circumference of the

gut. The surface of the growth is regularly and shallowly ulcerated. It is evident from the specimen that the lumen has been almost completely occluded.

There is some pouching of the mucosa behind the proximal ring of the growth. The mucosa above and below the growth appears normal, with regular well-formed valvuli conniventes. The muscularis proximal to the growth is considerably hypertrophied (thickness above, 0·4 cm.; thickness below, 0·2 cm.). The serosa is grey and glistening, except in the

FIG. 372.—*Case* 2. Radiograph of the abdomen after barium enema, showing the segment of intestine above the neoplasm with its characteristic (transverse striation) appearance due to obstruction.

region of the growth, where it is raised irregularly by pearly discrete and confluent nodules of subserosal growth. There is one small gland in the attached mesentery with a soft greyish cut surface.

Microscopical.—A well-formed adenocarcinoma with no apparent involvement of the associated lymphatic gland (*Fig.* 373).

Case 3.—This patient, a young man of 26, was admitted under the care of Professor Graham Simpson in April, 1927, with a history of three months' flatulence and abdominal pain. He was pale and had lost weight, being afraid to eat because this made him sick and brought on the pain shortly afterwards. The bowels had been constipated for some months.

An interesting point in his history was that he had been in the hospital the previous year with an acute Meckel's diverticulitis. No abnormality of the jejunum was detected at that period. Retarded emptying was the only unusual finding with barium-meal examination of the stomach and duodenum, and clinically there was some epigastric tenderness.

AT OPERATION.—Laparotomy revealed three large ulcers in the first part of the jejunum, the first being situated just distal to the duodenojejunal flexure. These ulcers were causing a stricture of the bowel at that level. There was no enlargement of the abdominal glands.

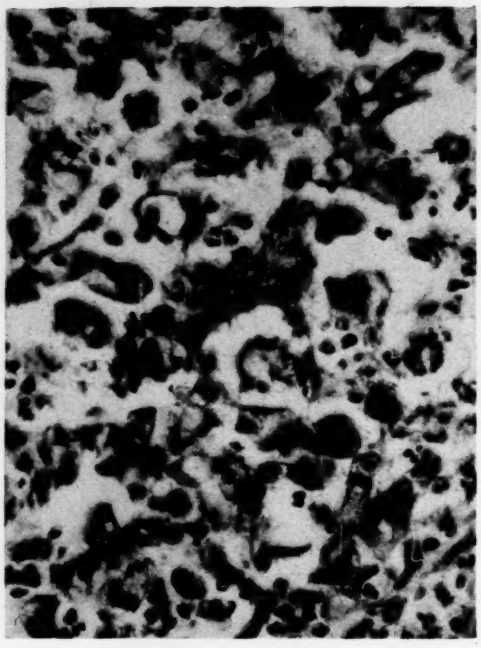


FIG. 373.—*Case* 2. Section of tumour.

A low gastrojejunostomy was performed, and this was successful in relieving the patient's symptoms for several months. The patient, however, became gradually weaker and signs of venous obstruction and secondary growth began to appear. A second laparotomy revealed that the jejunum, transverse colon, and omentum in the upper abdomen were matted together by malignant infiltration, further surgical treatment being impossible.

POST-MORTEM EXAMINATION (January, 1928).—There was considerable œdema of the ankles and scrotum, and marked abdominal distension due to ascites. Two pints of yellow turbid fluid were present in each pleural cavity. The glands of the mediastinum were enlarged, and were pressing on the root of the lung on the right side. The stomach, duodenum, jejunum, and omentum were matted together, forming a large mass of malignant tissue which extended towards the portal fissure. There was no evidence of ulceration in the stomach or large intestine, but small nodules of secondary growth were found on the peritoneal surface of the transverse and descending colon. The glands at the bifurcation of the aorta were enormously enlarged, white, and firm, and completely surrounded it on all sides. The spleen, suprarenals, kidneys, bladder, prostate, and testes were normal, but the liver contained a small mass of secondary growth the size of a shilling, on the upper aspect of the left lobe.

Microscopical.—The neoplasm was an adenocarcinoma.

DISCUSSION

The treatment of jejunal cancer with acute obstruction was discussed by d'Allaines, who, stressing the gravity of the condition, remarked that simple jejunostomy with its mortality of approximately 75 per cent was consequently of little value. Johnson made jejunostomy his operation of choice, but insisted that anastomosis should be performed within fourteen days in order to combat the rapid emaciation and the damage to the skin of the abdominal wall which is so likely to occur.

The severe effects of acute intestinal obstruction without strangulation can be attributed to a combination of factors. Most important is the alkalosis from loss of fluids and chlorides, due to fluid extravasation into the obstructed gut, and vomiting. Probably bacterial toxins and toxic metabolites formed in the intestinal wall also play a significant role. Clinically the loss of sleep and the anxiety resulting from incessant vomiting are seriously detrimental features of the condition.

If a diagnosis of high intestinal obstruction without strangulation could be made with certainty before operation, the correct line of treatment would clearly be the administration of continuous intravenous drip saline, combined with continuous pernasal duodenal aspiration, until the effect of the acute obstruction had subsided. Haden and Orr showed that in dogs with duodenal obstruction, life could be prolonged for several weeks simply by the administration of normal saline.

At this Hospital a number of cases with paralytic ileus (whether due to acute peritonitis or surgical anastomosis or other operation of the intestine) are being treated by duodenal aspiration. The results are proving distinctly gratifying, as evidenced clinically by rapid loss of nausea, improved sleep, and diminished abdominal distension.

Usually carcinoma of the jejunum which results in acute obstruction is discoverable only at operation. In these circumstances, if healthy intestine is present at the proposed site of anastomosis, resection followed by continuous pernasal duodenal aspiration and drip saline infusion would appear to offer good prospects of success. When the neoplasm is detected in the course of laparotomy, the details of surgical intervention adopted depend on the site and operability of the tumour. Resection of the neoplasm with the associated glands and mesentery seems to be the procedure of choice. Alternatively there may be performed side-to-side anastomosis, gastro-enterostomy, or anastomosis-in-Y by implanting the jejunum into the stomach and the duodenum into the jejunum.

Gosset reports on the beneficial effect of deep X rays. A tumour for which an anastomosis had been performed was found at laparotomy two months later to be almost completely destroyed by this treatment. The prognosis of carcinoma of the jejunum is, however, uniformly bad, life seldom being prolonged for more than three years after the onset of symptoms.

SUMMARY

1. The general characteristics of carcinoma of the jejunum are discussed from the clinical and pathological viewpoints, and illustrative cases quoted.

2. A case is described of acute intestinal obstruction with imminent perforation due to ring carcinoma of the jejunum in a man 27 years of age.

3. The treatment in general of high intestinal obstruction is considered, emphasis being laid on the value of continuous pernasal duodenal aspiration, combined with continuous intravenous drip infusion of normal saline.

I am greatly indebted to Prof. Graham Simpson, Mr. Ferguson Wilson, and Dr. C. G. Imrie for kindly giving me permission to publish these cases.

REFERENCES

MAYO, C. W., and NETTROUR, W. S., *Surg. Gynecol. and Obst.*, 1937, lxv, 303.
PLUNKETT, J. E., FOLEY, M. P., and SNELL, A. M., *Canad. Med. Assoc. Jour.*, 1936, xxxiv, 289.
JOHNSON, R., *Brit. Jour. Surg.*, 1921–22, ix, 422.
D'ALLAINES, F., *Jour. de Chir.*, 1929, xxxiii, 449.
BLAND-SUTTON, Sir JOHN, *Lancet*, 1914, ii, 931.
HEGGS, F. MITCHELL, *Lancet*, 1937, ii, 1250.

TWO CASES OF SARCOMA OF THE SMALL INTESTINE WITH UNUSUAL FEATURES

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I. LEIOMYOSARCOMA OF THE ILEUM

LEIOMYOSARCOMA in the small intestine is a rare tumour, if the published cases are a criterion of its frequency. A search of the literature reveals that only 30 cases have been described in detail.

CASE REPORT

HISTORY.—A female, aged 56, was admitted to hospital on May 14, 1937, complaining of colicky abdominal pain of 5½ months' duration, which was aggravated by food and often accompanied by vomiting. She had lost 3 st. in weight during the previous twelve months. There was no history of constipation, diarrhœa, or melæna, but she stated that she vomited blood three years before admission. She noticed that her finger-nails began to change shape and to split easily two months before admission.

ON EXAMINATION.—She was thin and pale, with slightly furred tongue. There was a hard, movable, slightly tender mass in the right iliac fossa, which was also palpable through

FIG. 374.—Cross-section of leiomyosarcoma of small intestine, showing cavity communicating with lumen of intestine.

the rectum, lying above and to the right. The liver was not enlarged. Her finger-nails showed the typical changes occasionally found in hypochromic anæmia—i.e., they were spoon-shaped and brittle. Blood-count on May 15, 1937, showed: red cells 3,100,000 per c.mm., hæmoglobin 42 per cent, colour index 0·7; white cells 8000 per c.mm., polymorphonuclear cells 76 per cent, lymphocytes 21 per cent, monocytes 3 per cent. The red cells showed slight anisocytosis and poikilocytosis. The mean corpuscular volume was 75 c.μ. (normal 80 to 95 c.μ). Occult blood-test, positive. Barium enema, normal. A test-meal showed free HCl.

During the period before operation she had a daily rise of temperature up to 99·5° F., with a pulse-rate of 70–80.

AT OPERATION.—On May 31, 1937, under nitrous oxide, oxygen, and ether anæsthesia, the abdomen was opened through a right lower paramedian incision. A hard, smooth, lobulated tumour, about the size of a fœtal head, was seen in the right iliac fossa, growing from the mesenteric border of the ileum into the mesentery, 12 in. from the ileocæcal valve.

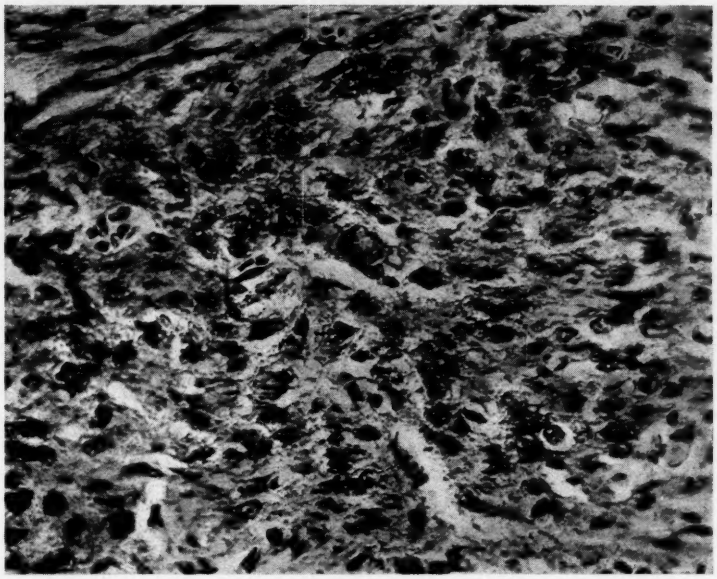

FIG. 375.—Leiomyosarcoma of ileum, showing multinucleated cells and hyperchromatic nuclei.

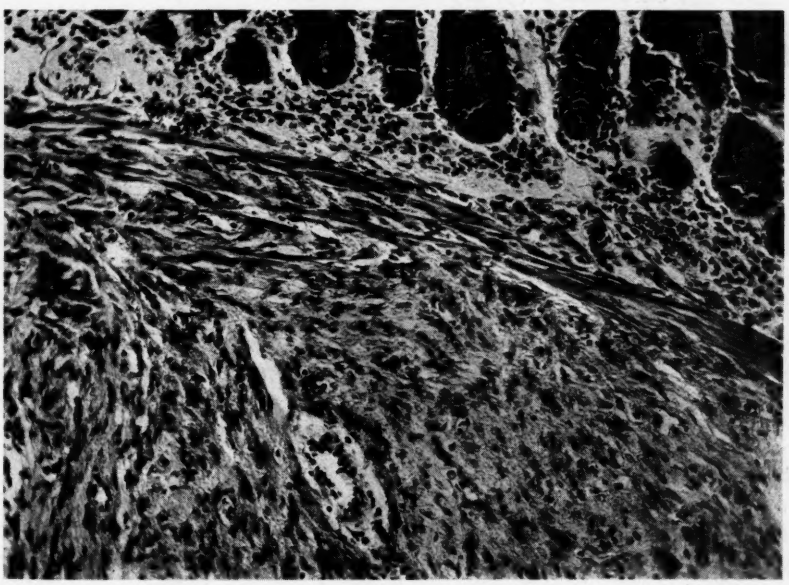

FIG. 376.—Leiomyosarcoma of ileum, showing tumour cells replacing the muscular coats.

There were a few fibrous adhesions between the growth and the uterus and peritoneum. The portion of small intestine containing the tumour was resected without difficulty, and a side-to-side anastomosis performed. Convalescence was uneventful. The blood-count

improved steadily, with the help of iron; and the finger-nails gradually become more normal in appearance. Six weeks after operation the blood-count and finger-nails were normal.

PATHOLOGICAL REPORT (Dr. Day, Central Histological Laboratory, Archway Hospital).—The specimen consists of a portion of small intestine 17 cm. long, which has been opened along the antimesenteric border. Expanding into the mesentery and occupying about half the circumference of the gut for a distance of 8 cm. is a tumour mass measuring 15 × 9 × 7 cm. Within the mass is an irregular cavity about 9 cm. in diameter, which communicates with the lumen of the intestine by a stoma 3 cm. in diameter (*Fig.* 374). The wall of the cavity is composed of soft greyish-white faintly lobulated tissue. On the back of the specimen, except for the cut surface of removal from the mesentery, the mass is entirely covered by the mesenteric peritoneum.

Microscopically the tissue composing the tumour is abundantly cellular, the cells being elongated and spindle shaped and having a fairly abundant eosinophil cytoplasm. The nuclei are hyperchromatic, and multinucleated cells are common (*Fig.* 375). Collagen fibrils are for the most part scanty. The tumour has replaced the middle coat of the ileum, but shows no tendency to infiltrate the peritoneum (*Fig.* 376). At the junction of the lumen of the gut and the cavity within the tumour the mucosa is atrophied and there is ulceration, but invasion of the submucosa by tumour cells is slight. A lymphatic gland lying in the mesentery close to the tumour showed no invasion.

On the basis of the presence of multinucleated cells, hyperchromatosis, and its cellularity, it was decided that the tumour was sarcomatous. In spite of the absence of myoglia fibrils when stained by Mallory's phosphotungstic acid hæmatoxylin, the general shape and arrangement of the constituent cells strongly suggest a myomatous origin. The absence of evidence of infiltration and the infrequency of mitotic figures indicate a favourable prognosis.

DISCUSSION

Ewing[1] divides sarcomas of the small intestine into the following groups: (1) Spindle-celled sarcomas, arising from unstriped muscle (leiomyosarcoma); (2) Miscellaneous sarcomas; (3) Lymphosarcomas.

Brink and Laing[2] say that 250 sarcomas of the small intestine have been recorded and of these approximately 6 to 8 per cent are myogenous.

Examination of the histories of the 30 recorded cases shows a somewhat similar symptomatology. The commonest symptoms are the passage of tarry stools and anæmia (14 cases). Pain is usually a late manifestation, is colicky in nature, and aggravated by food. This history often leads to the diagnosis of gastric or duodenal ulcer, although barium radiographs are negative. Persistent positive occult-blood tests, together with normal X-ray findings in the stomach and duodenum, should exclude an ulcer in this region. Other common symptoms are diarrhœa and loss of weight. Vomiting very rarely occurs, as these tumours do not cause obstruction.

The commonest clinical sign is the presence of a *movable* abdominal mass. This, together with a history of bleeding from the bowel, should suggest the possibility of a leiomyosarcoma of the small intestine. There may be a slight irregular temperature and leucocytosis.

X-ray examination is of little use in diagnosis unless there is ulceration of the mucosa with cavitation. When this occurs Lingley[5] has shown that it is possible to demonstrate the cavity by means of the barium which is left adherent to the walls after the main mass of barium has passed.

These tumours are usually found in the jejunum. Of 14 cases reported by Anderson and Doob,[6] 10 were in the jejunum, 3 in the duodenum, and 1 in the ileum. Subsequent reports confirm this incidence. The tumour varies in size from a walnut to a baby's head.

Complications.—One case each of perforation (Pugliatti)[3] and intussusception (Weeden)[4] have been described.

Secondary tumours are not common, as they were found in 6 cases only. The primary and secondary tumours grow slowly. One case gave rise to a severe melæna five years before death occurred from a similar attack. One patient developed secondary deposits thirteen years after successful removal of the primary growth.

The presence of spoon-shaped, brittle nails is interesting, as this is a common accompaniment of hypochromic anæmia from whatever cause. Tumours of the intestine may give rise to hypochromic anæmia as a result of continuous oozing of blood from the ulcerated surface.

Pathology.—These tumours arise from the muscular coats and produce large well-circumscribed solid or cystic growths. They are usually nodular, lobulated, yellowish-white in colour, and prone to degeneration in the centre. An early case sometimes lies external to an intact mucosa, but later it ulcerates through the mucosal surface. This occurred in about half the cases. The majority remain localized and grow outwards from the bowel into the mesentery, so that obstruction is very rare. The overlying peritoneum is usually normal in appearance.

The diagnosis of the malignancy rests upon the histological appearance of the tumour, as macroscopically it may resemble a leiomyoma. Ewing describes the cells as being larger than those of a leiomyoma, with more massive and hyperchromatic nuclei, and evidence of mitosis. The intercellular connective tissue is scanty. That the tumour arises from muscle cells is shown by the bundle arrangement of the cells and the presence of myoglial fibrils on staining with phosphotungstic acid hæmatoxylin.

II. LYMPHOSARCOMA OF THE SMALL INTESTINE WITH PERFORATION

Only 6 cases of perforation of a lymphosarcoma of the small intestine have been described in detail in the literature,[7-12] and as nearly 400 cases of lymphosarcoma of the intestines have been reported, perforation may be regarded as a rare complication. A better idea of the relative frequency of lymphosarcoma is given by Staemler[13] in an analysis of 54,000 autopsies in the German and Austrian hospitals in 1923. He found 33 cases of lymphosarcoma of the intestines—an incidence of 0·06 per cent. None of these cases were complicated by perforation.

CASE REPORT

HISTORY.—Male, aged 69. Sudden attack of severe abdominal pain ten minutes after dinner, while lifting a weight. This occurred four hours before admission to hospital. The pain came on suddenly, was continuous, and was accompanied by vomiting. There was no previous history of indigestion or illness.

ON ADMISSION.—He was very pale and shocked, with pulse-rate 144, temperature 97° F., and respirations 28 per minute.

ON EXAMINATION.—The tongue was moist and only slightly furred. There was generalized abdominal rigidity and considerable distension. No audible peristalsis. Liver dullness not diminished. Slight dullness in flanks. He vomited some dark brown fluid shortly before operation. The diagnosis of perforated gastric ulcer was made.

AT OPERATION.—As the patient was in poor condition, operation was performed under local anæsthesia, through a midline supra-umbilical incision. On opening the peritoneum

a large quantity of dark, blood-stained fluid escaped. Flakes of lymph were to be seen adherent to the small intestine. No ulcer was found in the stomach or duodenum. A hand passed down towards the pelvis encountered a flabby sac-like structure attached to the jejunum, 4 in. beyond the duodenojejunal flexure. This was easily drawn out of the incision and proved to be a saccular aneurysmal-like dilatation of the anti-mesenteric border of the jejunum. The wall of the sac was $\frac{1}{8}$ in. thick and was perforated at one point. The sac was filled with blood-clot, and altered blood was found in the jejunum beyond. No secondary deposits were seen in the glands of the liver. The affected portion of gut was

FIG. 377.—Lymphosarcoma of jejunum, showing aneurysmal dilatation of intestine and perforation.

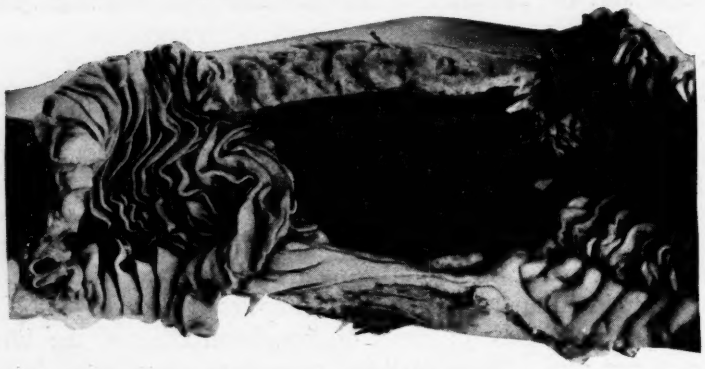

FIG. 378.—Lymphosarcoma of jejunum. On looking directly into the cavity of the tumour the perforation can be seen as a light triangular area.

resected and an end-to-end anastomosis performed. The blood-stained fluid was removed from the peritoneal cavity by aspiration, and the abdomen closed. The pulse gradually became weaker and death occurred six hours after operation.

PATHOLOGY OF SPECIMEN.—

Macroscopical Appearance (*Figs.* 377, 378).—The diverticulum measured 5 × 3 × 4 in. The outer surface was smooth and pink in colour, and covered with peritoneum. The inner surface was finely granular and gelatinous in appearance, merging rather abruptly with the normal mucous membrane. The perforation was triangular in shape and was situated slightly proximal to the greatest curvature. The wall of the tumour was soft and pliable and measured $\frac{1}{8}$ in. in thickness. The sac was filled with fresh clotted blood.

Microscopical Appearance.—The tumour is formed by closely packed cells. These are mostly large and small lymphocytes having scanty protoplasm and deeply stained nuclei

(*Fig.* 379). Near the middle of the tumour they completely replace the intestinal wall. The tumour infiltrates from without inwards, so that at its borders the muscularis and submucosa are infiltrated and largely destroyed beneath an intact mucosa (*Fig.* 380).

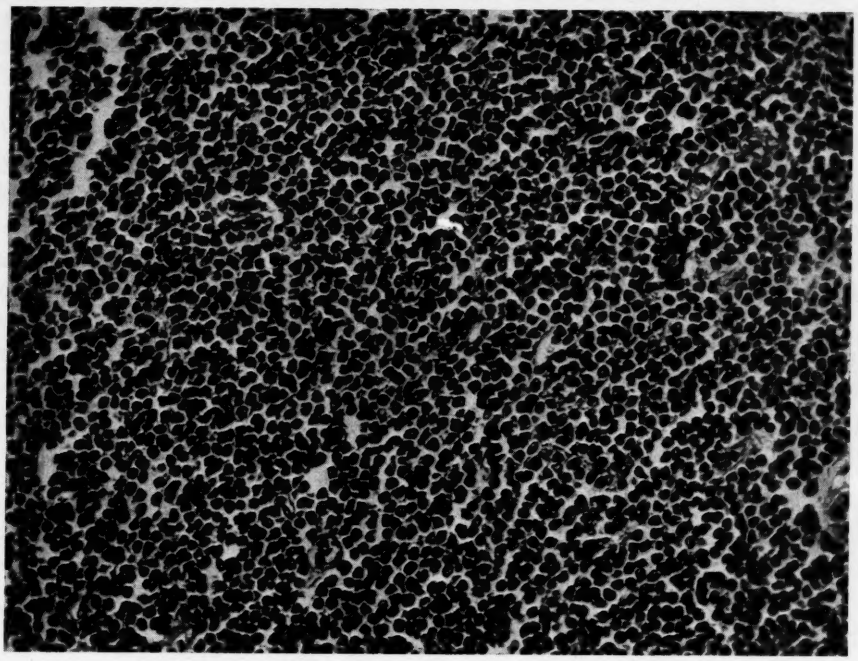

FIG. 379.—Lymphosarcoma of jejunum, showing the tumour composed of closely packed lymphocytes.

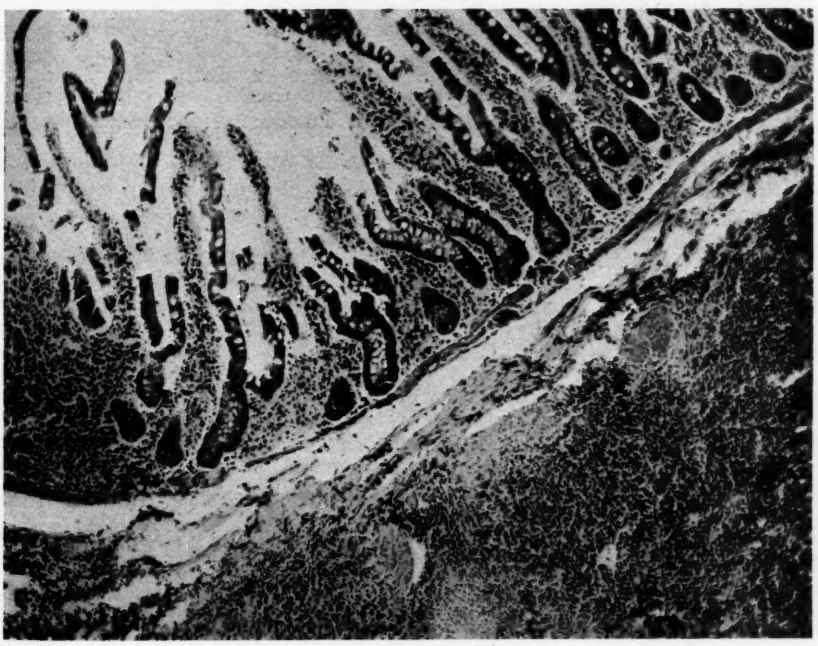

FIG. 380.—Lymphosarcoma of jejunum, showing infiltration of muscularis and submucosa.

POST-MORTEM FINDINGS.—A small subperitoneal plaque ½ in. long was found in a loop of transverse colon which was lying in contact with the tumour. It was not adherent and the overlying peritoneum was intact. Several tiny nodules, about the size of a pin's head, were present in the peritoneum at the root of the mesentery of the affected portion of the gut. These deposits had the same microscopical appearance as the primary growth. No other secondary deposits were found. The anastomosis was secure. Diffuse fibropurulent peritonitis was present.

DISCUSSION

Ullmann and Abeshouse,[14] in a review of 125 cases of lymphosarcoma of the intestines, found a tendency to aneurysmal dilatation in the majority. The probable explanation is that the growth spreads from the lymphoid follicles along the submucosa and muscularis mucosæ into the muscular layers. The dilatation may be due either to the infiltration of the muscular layers or to early destruction of the nerve plexuses in the submucous layer.

It is rather difficult to decide whether the hæmorrhage caused the perforation or whether the hæmorrhage was a secondary event arising from the ruptured area. The patient stated that the pain came on suddenly during effort, which suggests that the perforation occurred first and was due to pressure.

The photographs were prepared by Mr. G. W. Moore, senior technician at the Central Histological Laboratory, Archway Hospital, London.

REFERENCES

[1] EWING, J., *Neoplastic Diseases*, 3rd ed., 1928. Philadelphia and London: W. B. Saunders Co.

[2] BRINK, J. R., and LAING, G. H., "Leiomyosarcoma of the Jejunum", *Arch. of Pathol.*, 1933, xv, 316.

[3] PUGLIATTI, V., "Leiomioma sarcomatode diverticolare dell Ileo, a Esito insolito", *Clin. ostet.*, 1934, xxxvi, 702.

[4] WEEDEN, W. M., "Lymphosarcoma of the Gastro-intestinal Tract", *Ann. of Surg.*, 1929, xc, 247.

[5] LINGLEY, J. R., "Non-obstructing Malignant Tumours of the Small Bowel", *Amer. Jour. Roentgenol.*, 1936, xxxvi, 902.

[6] ANDERSON, D. H., and DOOB, E. F., "Leiomyosarcoma of the Duodenum", *Arch. of Pathol.*, 1933, xvi, 795.

[7] HULBERT, "Specimen of Sarcoma of Viscera", *St. Louis Med. and Surg. Jour.*, 1885, xlviii, 250.

[8] PISSAREWA, T., "Lymphogranulomatosis of the Digestive Tract", *Arch. f. klin. Chir.*, 1927, cxlix, 75.

[9] OETTLE, E., "Primary Sarcoma of Small Intestine as Cause of Perforating Peritonitis", *Deut. Zeits. f. Chir.*, 1935, ccxlv, 75.

[10] PREMOLI, F., "Peritonitis from Perforation of Sarcoma", *Rev. Med. del Rosario*, 1931, xxi, 458.

[11] DAVIES, E., "Lymphosarcoma with Perforation of Gastric and Intestinal New Growth", *Brit. Med. Jour.*, 1937, ii, 64.

[12] WHITAKER, L., and FISHER, J. H., "Primary Lymphosarcoma of Ileum with Perforation and Death from Acute Peritonitis", *Canad. Med. Assoc. Jour.*, 1934, xxx, 420.

[13] STAEMLER, M., "Neoplasms of the Intestines", *Deut. Chirurgie* (ed. G. Kuthner), 1923, xlvi, 296. Stuttgart.

[14] ULLMAN, A., and ABESHOUSE, B. S., "Lymphosarcoma of the Small and Large Intestines", *Ann. of Surg.*, 1932, xcv, 878.

ADAMANTINOMA OF THE TIBIA

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THIS case of adamantinoma of the tibia is considered worthy of record as so few cases have so far been reported.

Clinical examination and X-ray evidence have not in the majority of cases given rise to the diagnosis, and a histological examination of the tumour has been necessary before its true nature has been found.

There are many similar features present in the 5 cases recorded in the literature. The tumour is found in males and females alike. No particular occupation seems to predispose to the disease, and the age incidence reported varies from 24 to 46 years. The chronicity of the tumour is a noteworthy feature: the duration between the discovery of the lesion and the subsequent resection or amputation varies from 10 months to 16 years, with only local extension.

In all cases there has been a definite history of injury—the tumour has followed the alleged trauma, and is found at the site of the injury. It is this constant history of trauma which has given rise to the theory that epithelial cells of primitive basal-celled type become detached and implanted in the periosteum, and owing to a peculiar stimulus differentiate them into enameloblasts and then into an adamantinoma. The tumour when established is said to remain limited to bone, because adamantinoma is embryologically a basal-cell growth, and all tumours of the basal-cell type have a limited kind of malignancy, with little ability to invade new type of tissue.

Another theory to account for the formation of these tumours is that they are produced from fœtal rests, trauma playing an essential part in stimulating the rest into growth. Ryrie, in his paper, dislikes the theory of hypothetical cell rests in positions where they are not adequately explained on embryological grounds, and believes that the tumour originates from epithelium implanted at the time of injury and that because of "thwarted repair" this epithelium ultimately passes over into tumour growth.

CASE REPORT

HISTORY.—J. K., a young girl aged 19, a book-binder by occupation, came to the Out-patients' Department of St. George-in-the-East Hospital on June 29, 1937, with the following note from her doctor: "Patient had a slip on a wet iron grating last night and fell down. The case is interesting for the swelling below the knee (? tibia) which has been there for some time. There is fluctuation present, but the strange thing is that there is very little pain."

The patient was admitted, and I saw her in the ward on June 30, 1937. On further questioning, the patient said she had noticed a lump at the top of the right shin at the age of 12, and that as a child she always seemed to be hurting it.

ON EXAMINATION.—A swelling was present at the upper end of the right tibia and a smaller swelling in the lower third of the same bone. The larger swelling felt cystic and was tender on palpation, and the patient complained of pain on flexing the knee and moving

the right ankle. There was no enlargement of the inguinal glands, and no areas of tenderness or of tumour formation were found elsewhere in the body. A tentative diagnosis of fibro-cystic disease was made.

The Wassermann and Kahn reactions of the blood were negative and the blood-picture was within normal limits.

Apart from the presence of a fair number of epithelial cells and calcium oxalate crystals, the urine contained no abnormal constituents. Bence-Jones protein was not present.

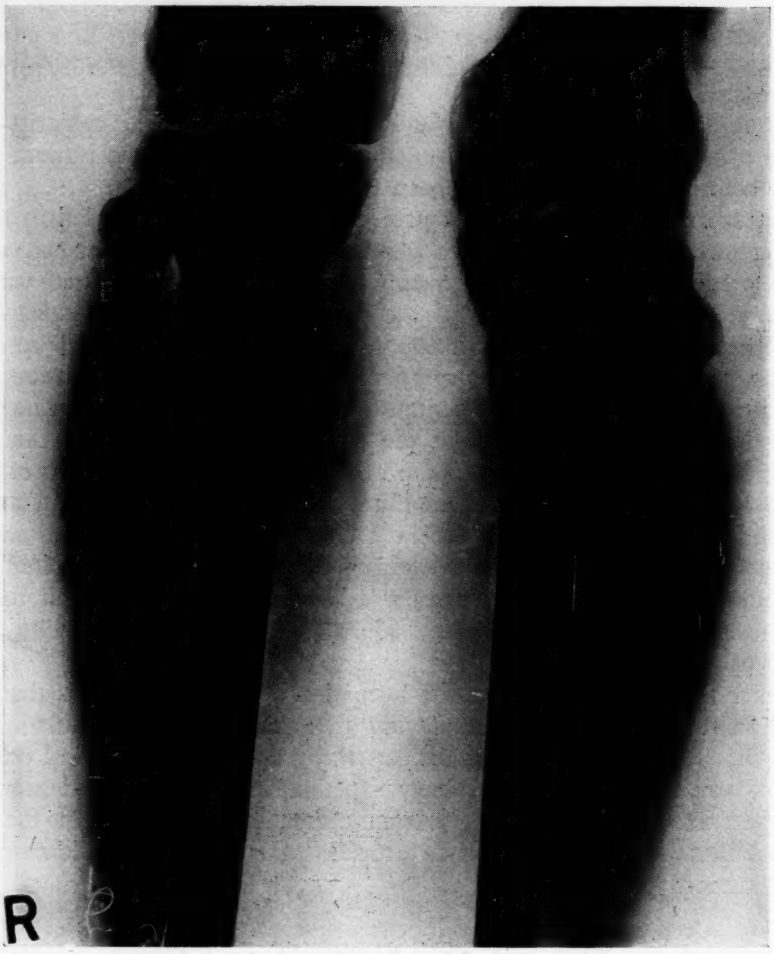


FIG. 381.—Radiograph showing the tumour in the upper end of the right tibia.

X-ray examination of the right tibia showed the presence of cystic changes, and suggested the presence of a giant-cell tumour of the upper end, possibly undergoing malignant changes, but the appearances were somewhat atypical (*Fig.* 381).

The patient's blood analysis was done by Dr. Allott, who reported as follows :—

July 6.	Serum calcium	9·6 mg. per cent
	Serum inorganic phosphate	3·7 mg. per cent
	Plasma phosphatase ..	0·23 units (Kay) (normal 0·1 to 0·22)

Calcium balance : 4-day period, July 10 to 15, 1937

		Calcium	*Phosphorus*
		g.	g.
	Output urine	1·48	2·05
	„ fæces	0·58	0·41
	Total output	2·06	2·46
	„ intake	0·40	2·0
	Balance for period ..	—1·66	—0·46
July 20.	Serum calcium	12·5 mg. per cent	
	Serum inorganic phosphate	3·4 mg. per cent	
	Plasma phosphatase ..	0·25 units (Kay)	

Dr. Allott wrote in a personal communication that "although the serum calcium was high and the calcium balance was a negative one, the plasma-phosphatase on two occasions

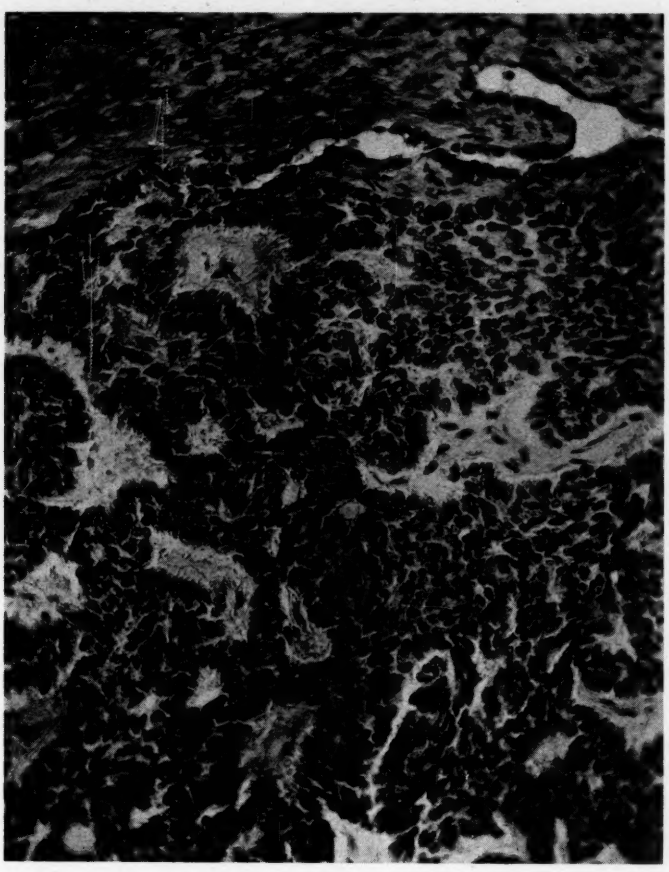


FIG. 382.—Microphotograph showing epithelial cells arranged in trabeculæ and solid masses, with columnar cells in the borders and a whorled appearance in the solid mass near the centre.

was only very slightly above normal, and this I think is probably against the diagnosis of fibrocystic disease".

TREATMENT.—

July 22, 1937.—The upper end of the right tibia was curetted for diagnostic purposes and the material obtained sent for histological examination. Sections taken were reported on by Dr. Barnard as "adamantinoma of tibia", and his report was as follows: "The

tumour consists of well-formed cells forming masses of strands separated by a loose connective tissue carrying blood-vessels. At the borders of the masses the cells are arranged in a palisade manner, and in the central part of some of them they form whorls. The cells themselves are mostly polygonal, spindle, or columnar, and have distinct oval nuclei with well-defined chromatin net and nodes. In the loose connective tissue there are in places numerous giant-celled osteoclasts, and near these there is old or recent hæmorrhage". (*Figs.* 382, 383.)

August 18, 1937.—An operation was performed by Mr. B. H. Burns. The cystic tumour on the upper end of the right tibia, as well as the involved medullary cavity as far as the

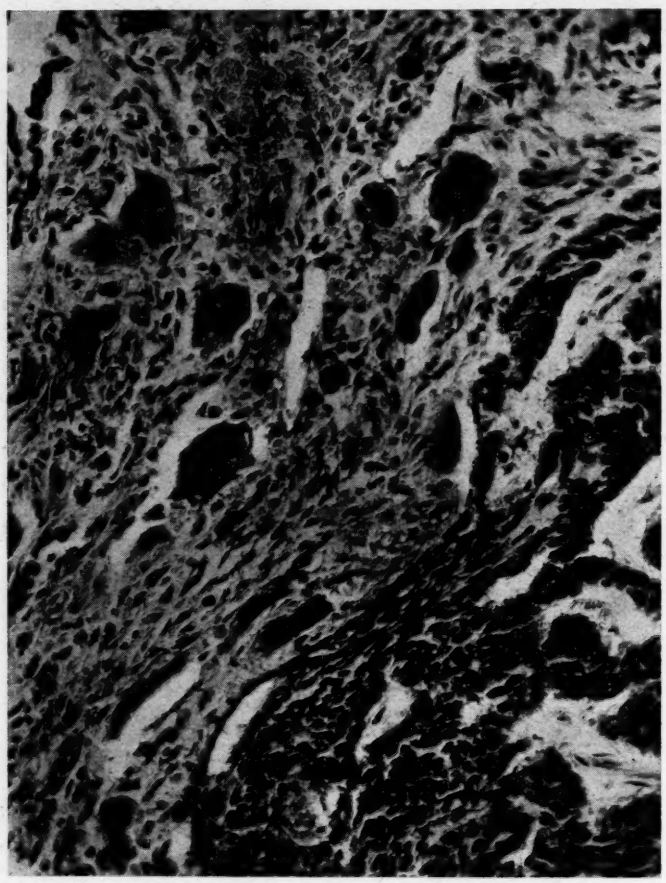


FIG. 383.—Showing loose connective tissue with hæmorrhage and giant osteoclasts.

smaller cyst in the lower third of the bone, were thoroughly curetted. The periosteum was found to be still intact, and in the upper third the corticalis was completely destroyed except on its anterior medial aspect. Two grafts taken from the left tibia were inserted into the whole length of the guttered right tibia. The limb was put in a plaster-of-Paris casing.

October 5, 1937.—The plaster was removed and a radiograph showed the bone-graft in good position (*Fig.* 384).

December 29, 1937.—There was no increase in the swelling of the leg. A further radiograph showed the graft in situ; there was little new-bone formation, but there was no marked absorption.

January 1, 1938.—By this time the patient was able to get about and bear a little weight on the leg, and she was sent to Ramsgate Convalescent Home. She returned on February 25,

and attended the out-patients' department. A radiograph showed the bone-graft in position, very little repair having occurred (*Fig.* 385).

The patient continued to attend the out-patients' department, and although she could get about quite well and complained of no pain, her general condition deteriorated and the swelling at the upper end of the right tibia increased, the soft tissues being now invaded.

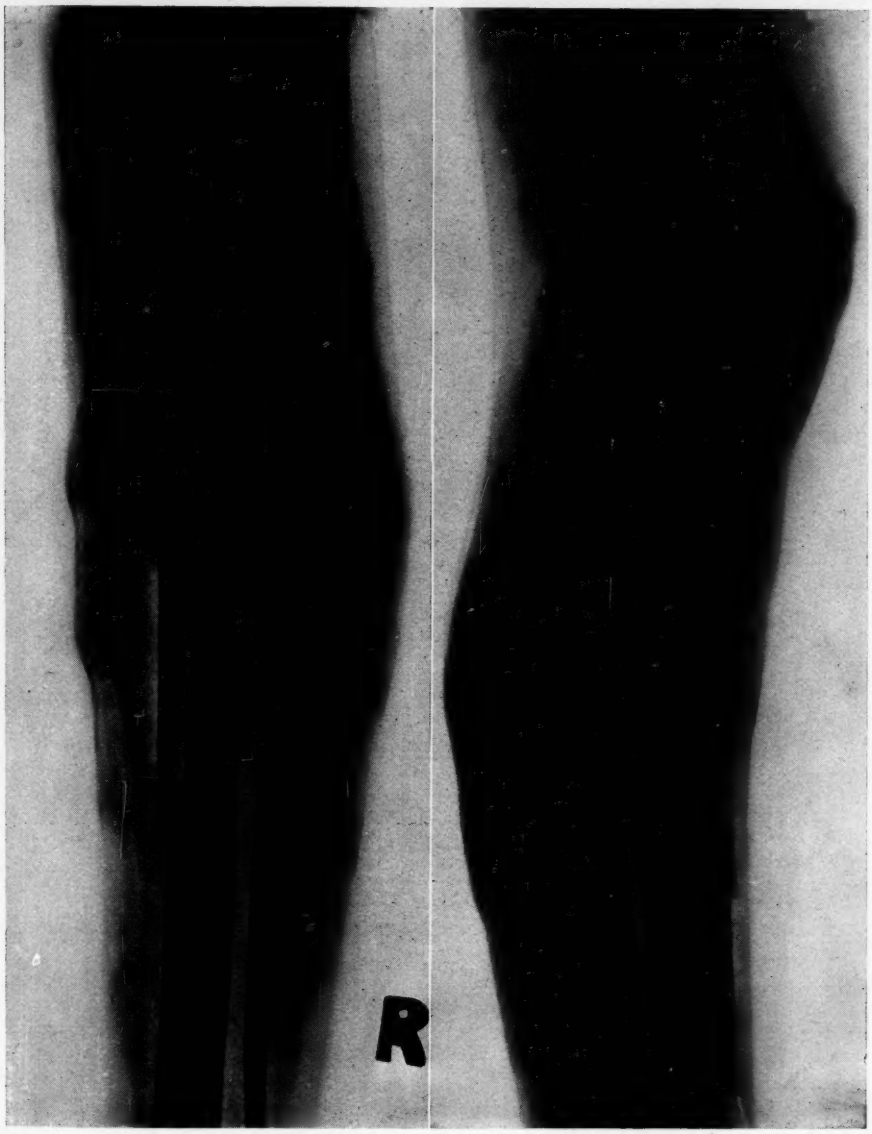


FIG. 384.—Radiographs showing the bone-graft in good position.

March 15, 1938.—She was re-admitted, and after consultation with Mr. B. H. Burns, it was decided to amputate the limb above the knee-joint. This was performed on March 28, 1938.

The amputated limb was sent to Dr. Barnard, who reported on the specimen as follows: "A right leg. Over the front of it and extending the full length of the tibia is a linear scar

which is puckered in one place 7 cm. from its upper end. The tibia is 34 cm. long and the lower 7 cm. consists of normal fatty cancellous bone and well-formed corticalis. Immediately above this level the cancellous bone is denser than usual and in it are two pieces of

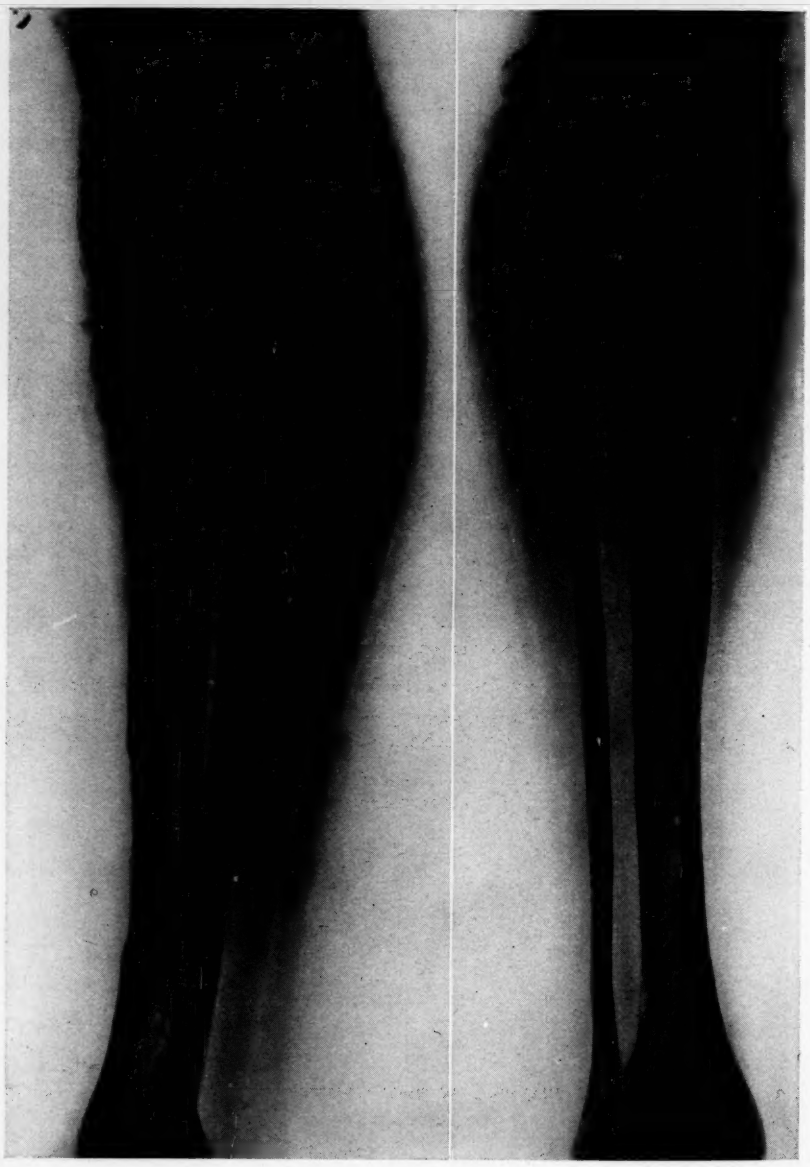


FIG. 385.—Radiographs taken more than four months later, showing the bone-graft in position, and the existence of but very little new-bone formation.

ivory bone each 5 cm. long and about 0·5 cm. thick. They are alined in the long axis of the bone with their adjacent ends slightly overlapping, and are nearer the anterior than the posterior surface. Over them the corticalis is represented by cancellous bone. There is a tumour 10 cm. long and 7 cm. wide occupying the whole of the shaft and that part of the

head 3 cm. below the articular cartilage. The tumour consists in part of cysts and in part of solid growth, the largest cyst being about 2·5 × 1·5 cm. The majority of the cysts are filled by soft grey and pinkish-grey growth. The solid part of the tumour consists of soft indistinctly lobulated grey, white, and pinkish-grey growth. In the central part of the growth ill-defined strands of cancellous bone can be identified. The whole tumour has a fairly well-formed capsule, but this has been infiltrated at its lower end." (*Fig.* 386.)

SUBSEQUENT PROGRESS.—The patient's general condition improved considerably after amputation and the wound healed by first intention. She was sent to a convalescent home on April 30, 1938, and she will be fitted with an artificial limb on her return.

FIG. 386.—Upper end of tibia, showing the character of the growth, and at the lower end the remains of the bone-graft. (× ½.)

SUMMARY

1. The tumour was present along the whole shaft of the tibia and invaded the soft tissues.

2. The case is another example of a localized tumour of bone giving rise to disordered calcium metabolism.

3. Radiation is said to have little effect on these tumours and was not used in this case. Curettage of the tumour and bone-grafting were tried, but amputation was found to be necessary.

I am indebted to Sir Frederick Menzies, Medical Officer of Health to The London County Council, for permission to publish this case, and to Dr. Leebody, Medical Superintendent of St. George-in-the-East Hospital, for his valuable assistance in preparing these notes.

BIBLIOGRAPHY

[1] BAKER, A. H., and HAWKSLEY, L. M., "A Case of Primary Adamantinoma of the Tibia", *Brit. Jour. Surg.*, 1931, xviii, 415.
[2] RYRIE, B. J., "Adamantinoma of the Tibia: Aetiology and Pathogenesis", *Brit. Med. Jour.*, 1932, ii, 1000.
[3] HOLDEN, EDGAR, and GRAY, J. W., "Adamantinoma of Tibia", *Jour. of Bone and Joint Surg.*, 1934, xvi, 401.
[4] BISHOP, EVERETT L., "Adamantinoma of the Tibia", *Southern Med. Jour.*, 1937, xxx, No. 6, 571.
[5] ALLOTT, E. M., and JENISON, J., "Generalized Osteitis Fibrosa due to a Retro-œsophageal Parathyroid Tumour", *Lancet*, 1938, i, 609.

NON-MALIGNANT STENOSIS OF THE ŒSOPHAGUS *

By G. GREY TURNER, London

"Let us cherish a sober mind, and take for granted that in our best performances there are latent many errors which in their own time will come to light . . ."—W. E. Gladstone, 1887.

I must confess that I have approached this subject with considerable hesitation, which has developed almost into trepidation as the time of the lecture has drawn nearer, for I cannot but recognize that I am perhaps trespassing on a field which belongs to another province of surgical practice. Nevertheless, I do feel that the subject is wrapped up with the recognition of general surgical principles and is of importance, not only from that point of view, but from its practical implications.

It is perhaps because of my life-long interest in that mournful Cinderella of surgery, malignant disease of the œsophagus, that I have enjoyed unusual opportunities for studying other lesions of the gullet which might otherwise have escaped my attention. I have always vividly remembered the picture of a non-malignant stricture in Walsham's *Surgery*: almost the first book on that subject which I acquired, now more than forty years ago, and which I still cherish. But experience has taught me that this type of stricture is very unusual, at least in this country. My first contact with an actual example of this condition occurred during the fruitful seven years which I occupied as Surgical Registrar at the Newcastle-on-Tyne Royal Infirmary. It was then my fortunate lot to make the post-mortem examinations on the patients who died in the surgical wards, and I cannot be too grateful for the very many valuable lessons which I learned. The case in question was that of a man of 35, who was admitted under the care of my great chief, Rutherford Morison. The poor fellow died of starvation, and I shall never forget going into the post-mortem room to make a last-minute examination, which had to be conducted in the coffin. Instead of the malignant growth of the œsophagus, which had been confidently diagnosed, the condition turned out to be a wonderful example of simple fibrous stricture at the lower end of the gullet, just as though a string had been tied around it. Quite possibly this resulted from the healing of an ulcer, for during the previous six years the man was said to have suffered from attacks of indigestion accompanied by vomiting, so that at times he could scarcely retain any food. For a month preceding admission he had only been able to swallow liquids, and for the previous two weeks had scarcely been able to get anything down. He was almost moribund on admission, and died eighteen hours after gastrostomy. Unfortunately, I have no further notes of the case, nor do I recall what became of the specimen, but the circumstances are indelibly fixed on my memory.

* This paper is based on a Presidential address delivered before the Section of Surgery of the Royal Society of Medicine, May, 1938.

Again, in the year 1901, I was much impressed by the story of a patient in the fifties on whom that careful and cultured surgeon, the late George Halliburton Hume, had performed gastrostomy under the impression that the patient was suffering from carcinoma of the œsophagus. The illness had been of only four months' duration and dysphagia was steadily progressive, with great loss of weight, so that the diagnosis appeared to have been amply justified. When I saw the man it was seven years after the operation, and he then turned up with the story that some few months after leaving hospital he had regained so much of the power of swallowing that he was able to get down enough soft food to satisfy his appetite. Being tired of the gastrostomy, he had allowed it to close, and since then had enjoyed good health and been able to carry on his not very laborious occupation.

FIG. 387.—Non-malignant stenosis of lower end of œsophagus. Patient, a woman of 66, had suffered from difficulty in swallowing for fifteen years. (*From St. Bartholomew's Hospital Museum, by permission.*)

Of course, these cases occurred before the days when it was possible to demonstrate these conditions by opaque drinks or with the œsophagoscope, and in diagnosis probability was allowed full sway. Nevertheless, the after-history of this particular case at least proved its non-malignant nature. But I soon learned that such cases were very exceptional, and for many years afterwards I saw no other example, and came to regard obstruction of the œsophagus in adults as almost synonymous with malignant disease. Nevertheless, I was very much struck by a casual remark which I heard Dr. W. J. Mayo make in his operating theatre in 1906, to the effect that it was always worth while doing gastrostomy because the diagnosis of cancer might be wrong.

But then, as now, the inquiring student searching our museums would find very few specimens of simple stenosis. There is a beautiful example in the museum of St. Bartholomew's Medical College (*Fig.* 387), and a very striking one in the Museum of the Royal College of Surgeons of Edinburgh (*Fig.* 388). The latter was placed there by Sir Charles Bell, and although it had been obtained after he had sought his fortune

in London, it was presented to the collection in his old school and remains a beautiful and interesting exhibit showing a fibrous stricture of many years' standing.

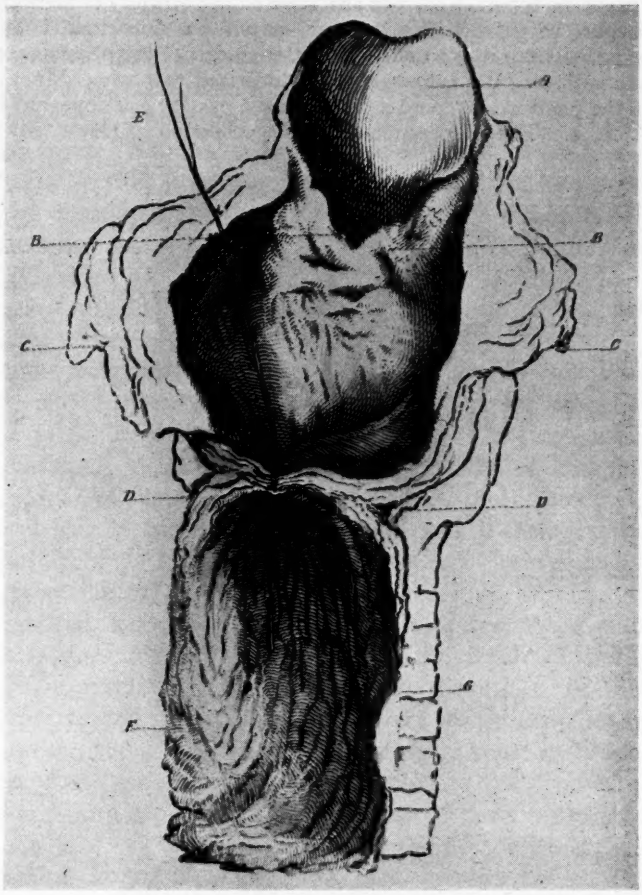


FIG. 388.—Cicatricial stenosis of upper œsophagus of twenty-four years' duration. ("*Surgical Observations*" *by Sir Charles Bell,* 1816–18.)

The description of the specimen, from Bell's *Surgical Observations* published in 1816 and 1818, is as follows :—

STRICTURE OF THE ŒSOPHAGUS FATAL

Drusilla Champ, resident in the parish of Bray, Berks, ascribed the commencement of her complaint to a cold and sore throat, which attacked her twenty-four years ago. After this she had difficulty of swallowing, but it was not attended to till the inconvenience becoming more considerable, in the year 1792, four years after its first occurrence, she made application to a surgeon. But as a superficial examination afforded no evidence of disease, very little was directed for her. She had an acid gargle, and a volatile liniment to rub her neck. After this she did not apply for medical assistance. The disease gradually increased, she still experiencing more and more difficulty in swallowing solid food of any kind, at length liquids produced great distress.

For the last ten years of her life it required an effort of many hours to swallow a small teacupful of thin fluid. On the 29th of May last she died, evidently from

starvation, occasioned by the impossibility of swallowing even fluid nourishment, sufficient to support life.

Hearing of the case, Mr. Goolden requested the husband to allow him to examine the part; he found (what you also have an opportunity of seeing), a stricture of the œsophagus, so narrow that it would admit no more than a horse-hair or bristle to pass; the stricture is situated at the lower part of the pharynx, and opposite to the cricoid cartilage. We cannot now be surprised that when Mr. Goolden put his finger into the pharynx, he could nowhere find a passage; it appeared a cul de sac. See plate IV. fig 1. The preparation is in the collection, XIII, 2. M. 8.

FIG. 389.—Fibrous stricture from scarring of high-lying gastric ulcer. The patient was a middle-aged woman who gave a long ulcer history.

Unfortunately there is no hint as to the age of the patient when the condition first started. My own lust for pathological specimens has, until quite recently, only added one example of non-malignant stricture to my collection (*Fig.* 389). This was an example of cicatrical stenosis of the lower end of the œsophagus following ulceration at the cardiac end of the stomach in a woman of 43. For years the patient had suffered from some sort of mental aberration and had been an anxiety to her parents. There was a long history of gastric trouble, with hæmatemesis on one occasion. She first began to have difficulty in swallowing two months before I saw her, affecting liquids more than solids. For the previous week she had taken little or nothing by the mouth and was very emaciated and feeble. An emergency gastrostomy was carried out, but the patient died of chest complications on the third day. The necropsy disclosed a fibrous narrowing of the lower end of the œsophagus leading to a definite stenosis just at the cardia, where there was the scar of a healed ulcer.

Although non-malignant stenosis is so uncommon in this country, it is not unusual abroad, and I have heard rumours of its great frequency in Russia, where it is said to follow attempts at suicide by swallowing strong alkalis. My friend, Alton Ochsner, of New Orleans, in a study of œsophagoplasty,[1] has collected the records of a large number of cases, and Guisez, quoted by Sir St. Clair Thomson and V. E. Negus in the last edition of their text-book on *Diseases of the Throat* published in 1927, states that of 25,000 cases of œsophageal disease diagnosed by direct vision through the œsophagoscope, no less than 185 were examples of cicatricial stenosis. Of these, 109 followed the swallowing of caustics, 7 the impaction of foreign bodies, 2 were the result of burns by very hot food, 5 resulted from war wounds, and 2 from the effects of toxic gases. But during recent years several cases have come before my notice showing the importance of this condition, and I want to refer especially to those in which it is assumed, possibly because of the age of the patient as much as the type of clinical history, that the condition is in all probability malignant. A striking example of this sort was that of a woman of 71, who was admitted under my care at the Hammersmith Hospital complaining of progressive dysphagia of twelve months' duration. The patient was sent to us with a diagnosis of malignant disease, and this appeared to be confirmed by a further X-ray examination. Nevertheless, the condition eventually turned out to be of a non-malignant nature. It may be well to record the history of this case, as it is especially instructive :—

Non-malignant Stenosis of the Upper Œsophagus in an Elderly Woman—Death from Mediastinal Abscess following Œsophagoscopy.—

HISTORY.—Until a year previously she had enjoyed good health and was the mother of five children. One day, while having breakfast, she suddenly found that a mouthful of food would not go down. The bolus was felt to pass to the back of the throat, but would go no farther and had to be ejected. The condition was progressive, and for the last two months she had noticed that fluids did not pass down so easily. Loss of weight and increasing weakness had occurred during the previous six months. She could swallow bread and butter, if well masticated, and the juice of meat, which was chewed and then spat out again. There was no pain or other complaint.

ON ADMISSION.—The patient was found to be a spare, feeble old woman, weighing only 4½ st. Her only complaint was of difficulty in swallowing. There were no external physical signs of any sort. In view of the very debilitated condition, it was decided to carry out a life-saving gastrostomy in the first instance, and to defer more detailed investigations until later. At this operation, on April 28, the absence of enlarged glands along the lesser curvature

FIG. 390.—Fibrous stenosis of upper œsophagus in a woman of 71. It was supposed to be malignant. Death from perforation, as indicated by the glass rod.

of the stomach or secondary deposits in the liver was noted. Recovery was very satisfactory, and the patient at once began to gain strength and weight, and at the end of a month was very much better and had put on 14 lb.

At this time a further X-ray examination was made, with report as follows: "Examination of the œsophagus shows an obstructive lesion at the commencement, causing a ragged filling defect in the œsophageal outline and much proximal dilatation of the pharynx. The stricture is not impermeable, a medium of the consistency of cream passing through quite easily."

On May 25 laryngoscopy was carried out. This showed the pharynx and larynx to be normal and without any suggestion of new growth.

Two days later the patient was submitted to œsophagoscopy under general anæsthesia, and this method of examination disclosed a very marked cicatricial narrowing just below the level of the cricoid cartilage. It required considerable manipulation to pass the œsophagoscope beyond this, but having passed the narrowed area the instrument was advanced with ease to its full length. No evidence of new growth was disclosed.

The patient was never quite well after this examination. She complained of sore throat and hoarseness and the neck was found to be tender. There was much mucus and difficulty in swallowing saliva. The pulse steadily rose and then the temperature. Death took place quite suddenly on the eighth day after the examination.

POST-MORTEM EXAMINATION.—At the autopsy the pharynx was found to be dilated down to the level of the cricoid cartilage, and at this point there was a stricture which had smooth, fibrous edges. Below this level the œsophagus appeared normal from the outside. Posterior to the strictured area there was an abscess cavity in the cellular tissues between the œsophagus and the spine, overlying the bodies of the last three cervical and first three dorsal vertebræ. There was a minute communication between the œsophagus at a point just above the stricture and the abscess cavity. The latter was filled with foul smelling pus. There was no evidence of disease in the underlying vertebral bodies. (*Fig.* 390.)

In both lungs there was evidence of old fibrosed nodules of tubercle scattered throughout the upper and middle lobes. There was also some basal bronchopneumonia with much œdema.

Microscopical examination of the œsophagus at the level of the stricture showed the epithelium intact and apparently healthy. The subepithelial layer was a little fibrosed and contained a number of macrophages, particularly just beneath the basal layer of epithelium. The muscular coat showed a fairly severe interstitial fibrosis separating the muscle fibres, and this was most marked at the point of the stricture. Outside this there was a layer of granulation tissue forming the wall of the pharyngeal abscess. There was no evidence of tubercle, syphilis, or new growth.

Comment.—It would appear possible that the œsophageal obstruction was due to a retropharyngeal inflammation followed by fibrosis of the muscle and that a retropharyngeal abscess was ruptured at the œsophagoscopy. On the other hand, there was nothing to suggest a cause for the abscess. In view of the long-standing history, it is more likely that the œsophageal stricture was the primary lesion.

My first introduction to the management of these conditions dates back to the year 1911, when I saw a good deal of a case treated by Professor Rutherford Morison in collaboration with the late lamented Hamilton Drummond. This case was published in *The Lancet*,[2] and I may just recall that the patient was a light-haired boy of 3 years of age, who had suffered from difficulty in swallowing from the age of 3 months. During the few months preceding admission he had become much worse, " vomiting " almost immediately after everything he took. The child was ravenous and always crying for food. His weight was only 20 lb. An X-ray photograph showed a stricture at the level of the upper border of the 10th dorsal vertebra. Attempts were made to pass a small tube into the stomach through the stenosis, but this not was not a success and the child continued to lose weight. In September of 1911, gastrostomy was carried out, and on October 7 an unsuccessful attempt was made to pass a ureteric catheter through the stricture from below, using a cystoscope through the gastrostomy opening. Further attempts at bougie treatment by the mouth and the use of a swallowed silk thread were also unavailing. As the result of the gastrostomy the boy rapidly improved and when he returned home he was just over 28 lb. in weight.

About a year later he was re-admitted for further treatment. He was then in good condition and looked like a normal child. With the œsophagoscope the gullet was found to be considerably dilated above a stricture, which was seen 26 cm. from the teeth. A fine whalebone bougie was passed through the stenosis into the stomach. This was followed by other bougies up to No. 8, which was left in situ for some hours. A fortnight later the stricture was seen to be dilated to about the size of a goose-quill. A No. 9 gum-elastic bougie was passed and left in situ for six hours. By December 12 a No. 15 bougie could be passed and this was again left in situ for two hours. In the interval the child had been taking food very well and was rapidly gaining weight. Bougies were now passed each week by the mouth without an anæsthetic. Soon afterwards the child was sent home, where his mother passed a No. 12 bougie without the least trouble.

In March, 1938, that is to say 25 years after completion of treatment in the Newcastle-upon-Tyne Infirmary, this patient reported as follows: Now thirty years of age, in good health, about 5 ft. 8 in. high, and weighing 9 st. 2 lb. Able to eat anything as long as well masticated (this was a life-long habit, as he had been taught to chew his food thoroughly as long as ever he could remember). The bougie had been passed regularly every day until he was 12 years old, then every other day until 14, and from that time until 18 years of age once or twice every week. From the latter age the bougie had never been passed and there had been no tendency to recurrence of the stricture. At the time of writing he was working regularly and had been married for three years.

In 1912 it was my good fortune to pay a memorable visit to Vienna as a member of the Surgical Club (now the Moynihan Chirurgical Club), and on that occasion we spent a delightful afternoon in the Clinic of Professor Lothiessen, where we were shown many examples of non-malignant stricture of the œsophagus,

mostly in children or young persons, and all I think due to the swallowing of corrosive fluids. In Vienna at that time a great deal of the washing, at least among industrial workers, was done in the homes of the people, as those of you will remember who knew that delightful city in the old happy days. One of my most vivid recollections is of the back of the houses as seen from a boat on the Danube, each with a long pole sticking out of every window on which was suspended the major part of the weekly wash, in many cases the most confidential garments fluttering in the wind! In order to get the clothes as white as possible, the housewives used caustic lye, and it appears that the children often got hold of this and, for some unexplained reason, frequently swallowed a quantity. Professor Lothiessen was treating these cases, not by passing bougies himself, but *by teaching the little victims to pass their own bougies*, and I shall never forget the sight of a dozen or more of these children sitting on wide shelves ranged round the room, carefully negotiating their strictures with bougies under the persuasive efforts of the kindly professor. He also showed us many cases on which the method had been carried out with great success, and I think we all came away convinced of its value. This was an experience which I have never forgotten and which has been of great value to me in dealing with the few cases of this kind that have come before my notice.

In this country at the present time the management of this condition is usually undertaken by those in charge of special throat clinics, and the standard procedure seems to be dilatation of the stenosis through the œsophagoscope under the guidance of the eye. Though I cannot doubt that many of the cases are successfully dealt with in this way, it has been my lot to have been consulted in several instances in which this standard method of treatment has partially or wholly failed. A review of these cases conveys a great many important lessons with which I propose to dealt

In the following case I was asked to see the patient on account of an acute septic illness, which had followed an attempt to dilate such a stricture through the œsophagoscope. It was because the boy unexpectedly recovered from this very serious complication that he was later referred to my care for the trial of other means of treatment. It was assumed that further dilatation was likely to be ineffectual as well as dangerous, and that something in the way of a radical operation, such as œsophagoplasty, would be required. The details unfold themselves in the record of the case which follows.

Fibrous Stricture probably of Congenital Origin—Acute Mediastinitis and Empyema after Dilatation through Œsophagoscope—Gastrostomy and Exploration of Lower Œsophagus—Relief finally Obtained by Method of Self-dilatation.—

HISTORY.—The patient (O'Hare, Newcastle-upon-Tyne), a lad of 19, was admitted under my care on May 22, 1934. He stated that as long as ever he could remember he had suffered from difficulty in swallowing, and he could recall his mother saying that he had vomited his baby's-bottle milk. During schooldays he could not take the meals provided, but had to rely on liquids and soft food. He could eat soft biscuits, cake, and suet pudding, but not meat or bread and butter. Even then he had good and bad days, and when at his worst could hardly swallow anything. He got some medicine from a doctor and thinks he improved a little.

In 1927 when he was 12 years of age, he first took serious advice and was seen by Dr. Horsley Drummond. He was X-rayed after an opaque drink and the œsophageal stenosis was confirmed. Treatment by Hurst's bougie was tried, but this did not pass the narrowing. About a week later, under general anæsthesia, bougies were passed through the œsophagoscope.

After this he could swallow ordinary food with ease, but this happy state lasted only for a short time, when the trouble recurred. After this he had intermittent difficulty, often worse at week-ends when he was at his own home.

By the end of a couple of years his diet had to be much restricted and he could not take meat and potatoes. Bougies were again passed and with some relief lasting for a few months.

After this his condition got gradually worse, and by the autumn of 1933 he was restricted to fluids, which he often regurgitated. He had difficulty in swallowing his own saliva and was losing weight. In September Hurst's bougie was again tried, but without success. Under general anæsthesia an attempt was made to carry out dilatation through the œsophagoscope, but this also failed, and the patient became very ill immediately afterwards.

It was at this stage that I was asked to see the case, and came to the conclusion that he was suffering from acute mediastinitis and would probably die. Fortunately the condition localized and a left-sided empyema formed and was drained, after which the acute symptoms gradually subsided. For about a month after this dilatation, which had been attended by such dire consequences, he could swallow much better, but the difficulty soon recurred, and by October gastrostomy became imperative. Following this operation swallowing improved a little, but again only for a short time. All this time the empyema was still discharging, but the condition slowly improved, and by the end of April of 1934 it had healed. The swallowing capacity had meanwhile got worse, and he soon arrived at a state in which he could hardly swallow his own saliva. A radiograph (*Fig.* 391) showed a long linear stenosis in the lower œsophagus. After deliberation I came to the conclusion that if mobilization of the lower end of the œsophagus could be carried out from the abdomen, it might be possible to perform cardioplasty, and accordingly the following operation was performed on May 25, 1934.

FIG. 391.—Fibrous stricture probably of congenital origin.

OPERATION.—The abdomen was opened by a long midline incision carried up into the angle between the ziphisternum and the left costal margin. In order to expose the œsophagus, the left lobe of the liver was mobilized and drawn over to the right. Signs of not very old inflammation were evident around the œsophageal hiatus, and there were some enlarged lymph-glands in the vicinity. The extreme lower end of the œsophagus could just be mobilized enough to enable the finger to encircle it, but above this point it was absolutely fixed and would not draw down, so that it was impossible to expose the whole extent of the narrowing. A bougie could not be passed through the stricture from the mouth and it was decided to attempt the retrograde route. An incision was made into the body of the stomach and the cardiac orifice explored by the finger from within; but as this could not be satisfactorily reached, the incision was closed and a vertical one made just below the cardia. Through this more conveniently situated opening the stricture was readily accessible, and was found to be definite and firm, with its lower limit about 2 in. above the diaphragm. As the lesion was too high up to be properly exposed, and as the œsophagus could not be mobilized, direct operative interference was considered to be out of the question. With considerable difficulty the opening in the cardia was eventually satisfactorily closed by catgut suture. The abdomen was closed, the gastrostomy tube being left in situ.

Comment.—I was doubtful about the outcome of the operation in this case for two reasons: (1) Because I considered that at the time the stricture was dilated, the bougie

had probably perforated the œsophagus and given rise to mediastinitis, and that, in consequence, the œsophagus would be fixed to the surrounding parts, and could probably not be drawn down towards the abdomen. (2) The appearance of the radiograph suggested that the stenosis commenced 2 to 3 in. above the diaphragm, which would be too high up to be safely reached for the purpose of a plastic operation. Both these points were verified at the operation.

In view of the disclosures of the operation, my plan was to suggest to the patient that the stricture should be left alone and that we should endeavour to make him a new antethoracic œsophagus.

SUBSEQUENT PROGRESS.—The patient made a very good recovery and swallowed just a little better afterwards, not only his saliva, but small quantities of water. In view of this encouraging improvement, I thought it proper again to attempt the passage of bougies with the assistance of the patient after the method of Professor Lothiessen. The whole proceeding was carefully explained to the patient and both Sister Strachan and Dr. Lachlan Fraser, my House Surgeon, co-operated very loyally and with much perseverance, and success soon attended their efforts.

At first only the very smallest bougies could be got through, but these were followed by rather larger ones passed daily, starting about the middle of June, and he was soon able to take fluid nourishment with ease. By the end of a month he was passing a little finger-size bougie and could eat bread, milk, and cake. Fluids went down quite easily and without any delay. He was able to leave hospital on July 2, taking with him a couple of bougies, which he passed each day. The capacity of swallowing gradually improved and with it his general condition. By early October he could pass a bougie of fore-finger size and by that time he could take ordinary food with his family and the gastrostomy was allowed to close. By early November he was very well, taking ordinary food and gaining weight rapidly. He was then only passing the bougie on alternate days.

In February of 1936, the patient visited the Newcastle-upon-Tyne Infirmary and saw Mr. Munro Black, who kindly reported to me as follows: "O'Hare is in splendid health, although still conscious of his disability. Owing to having his lunch away from home and wanting to feel sure that he is all right, he passes his large bougie about two or three times a week. When he passes the bougie, which he does without lubricant, he occasionally feels it a little tight at the lower end. On these occasions he passes it again or leaves it in for a short time. He looks very well and is of good weight. He eats any kind of food and does not require to exercise care in his diet. He is very pleased with the result of his treatment and does not consider the bougies any inconvenience."

In March, 1938, Mr. Munro Black again saw this patient and reported that: "He is very well in every way and able to eat normal food without any trace of trouble. He has passed his bougie, more or less just to keep his hand in, at about two-monthly intervals. X-ray examination shows a definite tapering narrowing in the lower œsophagus about 1 in. long, but only very temporary arrest of barium—a plate was not made. I have told him that he ought not to wait longer than a month without passing his bougie."

But all cases are not quite of this severe nature, and the next example I want to bring before your notice is that of a man whom I had the opportunity of treating in the first instance, and the brief record which follows shows how satisfactorily self-dilatation can be carried out and the lasting nature of the result.

Fibrous Stricture the result of Swallowing Ammonia—Dilatation by Self-passage of Bougies, with long-standing Relief.—The patient was a man of 57 (Williams), a nervous introspective fellow, often ailing and always complaining. In December of 1931 he became seriously depressed, and swallowed a quantity of strong ammonia; it was an old solution, but active enough to produce corrosion of his mouth and throat, and he had to be admitted to hospital for treatment. As the immediate effects wore off he developed dysphagia, and soon arrived at a state in which he could only swallow liquids with considerable difficulty. In February X-ray examination showed some narrowing just below the level of the cricoid, and it required several violent swallowing efforts to get the opaque material to pass. By April the obstruction appeared to have moved a little lower down and the upper end of the œsophagus was definitely dilated above it. Swallowing was a great trial, but he managed to

get enough liquid food down to keep up his habitual poor nutrition. He persevered without treatment until August, 1932, when he had to give in.

At this stage the patient was admitted under my care to a private hospital. At that time he was only able to exist on fluids, which he had to take very slowly and with great care. The object of the treatment by the use of bougies was carefully explained and I was fortunate in having a most painstaking and persevering matron who took the matter in hand. As the result of her efforts the patient soon learned to pass the bougies, and as the improvement was immediate, he carried this on assiduously. He remained in the home from August 17 until September 4, and by the time he left he was passing a bougie the size of the little finger before each meal. He could take ordinary food quite well and his mental condition had greatly improved.

At home his doctor passed the bougie once or twice and then the patient used it himself twice daily for some months, then gradually diminished the frequency. Its use was discontinued at the end of six months and he has never required to pass it again.

In March, 1938, Dr. James Waters reported that the patient continued in good health and could swallow quite naturally, though he stated that he never took a large mouthful.

The next case was also that of a patient in whom the traditional method had singularly failed, and it was assumed that nothing but an œsophagoplasty would meet the indications. It is therefore very interesting to note that the simpler and what may be looked upon as old-fashioned method again proved successful.

Fibrous Stricture the Result of Swallowing Ammonia – Failure of Dilatation through Œsophagoscope—Gastrostomy—Successful Self-dilatation.—The patient was a girl of 17 who drank some strong ammonia on March 23, 1934, and was treated at home. Difficulty in swallowing soon developed, and on April 27 the patient was admitted to the Throat Department of the Newcastle Infirmary on account of dysphagia both for fluids and solids. She was found to be suffering from inflammatory stricture at the commencement of the œsophagus and at the crossing of the left bronchus. These narrowings were dilated by bougies passed through the œsophagoscope. This caused bleeding, but was followed by some improvement. She was discharged on May 8, but was readmitted on May 20, as by that time she had again great difficulty in swallowing even liquids and was rapidly losing flesh. Œsophagoscopy carried out in the Throat Department showed a fibrous stricture commencing just behind the cricoid and extending downwards for 3 or 4 in., where the block became absolute. The notes stated that the condition was a fibrous stricture, but the upper end of the narrowing was in the midst of a dense mass of velvety granulation tissue. It was not possible to negotiate the narrowings under the guidance of the eye, and it was suggested that an antethoracic œsophagoplasty would probably be necessary and she was transferred to my care.

Operation.—As a first step, and to relieve the urgent dysphagia, gastrostomy was carried out, after which there was some little improvement in the swallowing capacity. The girl rapidly picked up and was able to leave the hospital on June 4.

On July 24 œsophagoscopy was again carried out and this confirmed stenosis, which appeared to be impassable, at 13½ in. from the teeth.

The patient was again readmitted on July 28, and as she was able to swallow some liquid, though slowly and with great difficulty, it was decided to make a trial of bougies passed by the patient herself. This treatment was carried out as in the other cases and with such promise that by the time the patient left hospital on August 14 she could pass a little finger-sized bougie and was able to swallow ordinary food. The gastrostomy was allowed to close. The patient reported again in October. She was then plump and well, and able to eat any sort of food and much more quickly than when she left hospital. She continued to use the bougie regularly.

Fourteen months later, that is, in February of 1936, she reported at the hospital once again, and was found to be a well-nourished, comely young woman, able to take ordinary diet without requiring to restrict her feeding in any way. About every two months she used her bougie, as by that time she felt the slightest suspicion of oncoming difficulty, which was immediately relieved by the passage of the instrument.

In March, 1938, the patient reported herself as very well. Two months previously she suffered from an attack of tonsillitis and was below par, but soon picked up. Just after this illness she fainted at work and when offered a drink found she was unable to swallow it. After passing her bougie, which she did with great ease, she found that she was able to swallow quite well. For several weeks previously she had neglected to use the bougie. The girl was leading a normal life and taking food without difficulty. She looked well nourished, weighed 8 st. 4 lb., and was cheerful and happy. As a precaution she was using the bougie once each day. By some strange freak, an X-ray examination has never been made in this case!

Even in elderly people dilatation with bougies can sometimes be carried out with success, and the next case I have to relate is of that nature.

Fibrous Stricture of Twenty-five Years' Duration in a Man of 81—Complete Relief by Self-dilatation.—The patient (R. T., Hammersmith Hospital, March, 1938) was a retired carpenter, 81 years of age, who was admitted complaining of dysphagia of 25 years' duration. The history was difficult to obtain accurately as the patient was rather deaf and his mind wandered to other subjects, but his daughter confirms that the account which he gave is substantially correct.

Even as a small boy certain foods, e.g., pork, used to be brought up after eating. Three to four years before the dysphagia started he had indigestion. The dysphagia was of gradual onset; it had not been continuous, but had come on at intervals. Between the intervals he could eat meat, if carefully chopped up and well masticated, but during the past five or six years he has not been able to do so. At the time of admission his diet consisted of rice, sago, eggs, milk, and tea, but these foodstuffs he took with increasing difficulty. During the last four years he had lost a great deal of weight.

During 1935 and 1936 he attended a London Hospital on account of the dysphagia, and the following report has kindly been supplied by the Registrar:—

"This man first attended in March, 1935.

"X-ray examination revealed constriction at the lower end of the œsophagus, extending for some distance above the diaphragm. Constriction was constant and did not dilate, the appearances therefore suggesting an organic, rather than a spasmodic, obstruction.

"Œsophagoscopy was performed on April 2, 1935, and this revealed leukoplakia at the lower end of the gullet, but there was no other lesion visible. The stricture was dilated. Œsophagoscopy was again performed on June 7, and the stricture dilated once more. Similar findings are recorded after œsophagoscopy on Feb. 10, 1936, and April 17. A section has not on any occasion been removed for microscopic examination."

On examination he was found to be a very thin, pale, wasted old man with organs generally exhibiting the changes incidental to advancing years. He was only able to swallow liquids. There were no ordinary physical signs of any condition likely to cause dysphagia. On February 2 a barium swallow was administered, and disclosed "obstructive lesion of œsophagus 5 in. above the level of the diaphragmatic hiatus, with gross proximal dilatation. Marked dorsal spondylitis." It was an ordeal for this enfeebled patient to attend the X-ray department, and for some days afterwards he was upset and delirious at night. After a few days he picked up, and on February 9 treatment by œsophageal bougies was begun, No. 15 being the largest that could be passed. The next day bougies up to No. 21 were used and were swallowed with comparative ease and little discomfort. The patient was very keen to try swallowing later in the day and managed some jelly and warm milk. The instruments were thereafter passed each day, and on Feb. 22 a further X-ray examination was made: "Compared with film on Feb. 5 there is a less degree of dilatation of the proximal gullet and an opaque medium of the consistency of cream passes with comparative ease through the stricture. The condition is much improved."

By March 4 he felt much stronger and looked forward to his food.

On March 11 the patient was obviously very much better. He looked almost fat in the face and was steadily gaining weight. He ate the ordinary diet of the ward and finished each meal, which he seemed to be able to take at about the same rate as the other patients. He was much brighter, more intelligent, and quite lively.

On April 20 the patient was very well and taking ordinary diet. He weighed 7 st. 3 lb., a gain of 10 lb. since treatment was started. Lately the bougie has only been passed once

a week, but is to be continued. By July the improvement was maintained and he had gained a further 9½ lb.

One of the most interesting of my cases was that of the man whose notes are to follow. It proved an eloquent example of the occasional futility of relying upon intermittent dilatation through the œsophagoscope, and it does show what the less dramatic method may sometimes attain in the way of successful treatment. The written record scarcely gives an indication of the psychological background of this particular patient. The man was a highly intelligent individual and his utterly hopeless attitude is difficult to realize except by those who came in contact with him at the time he was admitted to hospital. He had been repeatedly treated by dilatation through the œsophagoscope, and when it was suggested that we should once again try dilatation, he became very despondent and was reluctant to submit. As he was so intelligent, I allowed him to come to a lecture in which I had occasion to relate the circumstances of other somewhat similar cases that had responded to the treatment which was proposed in his case. The lecture was illustrated by lantern slides and was evidently enough to convince him that at least the method was worth a trial. That he did so well is a tribute to his confidence and perseverance.

Fibrous Stricture from Swallowing Hydrochloric Acid – Gastrostomy and repeated Dilatation through Gastroscope followed by Recurrence—Great Despondency—Eventual and Satisfactory Control by Method of Self-dilatation.—The patient (G. Y.) was a man of 29, who was admitted to the Hammersmith Hospital in August, 1936. He complained of dysphagia the result of swallowing hydrochloric acid four years previously. In 1931 he had some obscure illness with fever, said to be the result of heat-stroke. Following this he had fits of ague, but these were not found to be due to malaria. He then had pains in the right side, diagnosed as renal calculus. An operation was carried out, but the wound became septic and broke down several times. After this some fresh inflammation took place, with the appearance of a swelling in the right groin. Next year he developed appendicitis and was submitted to operation, the appendix being removed and a pelvic abscess drained. Two days later he developed pneumonia from which he did not recover for several weeks. Three months after the onset of the pneumonia, i.e., in August of 1932, he became very depressed, and it was then that he swallowed the acid and made an attempt to cut his throat. The acid was apparently a concentrated solution, as the patient noted acrid fumes before swallowing the fluid.

About seven days later he could not swallow his saliva, and it was then that gastrostomy was carried out. From September, 1932, to late in 1934 he took the main portion of his food by this route. Dilatation with gum elastic œsophageal bougies, passed once a week, was attempted, but this was not successful. Attempts were also made under general anæsthesia, but these also were unavailing. He was transferred to St. Mary Abbotts in February, 1933. Attempts were made to pass bougies with the aid of the œsophagoscope, but were not successful, and after a time the patient went to the North of England. Towards the end of 1933, in Liverpool, an " operation " was done in which an opening was established through the stricture. A secondary operation was also necessary. The opening was kept up by weekly dilatation. The gastrostomy was not allowed to close till about the latter end of 1934. He got on tolerably well in 1934 and was able to take semi-solids by the mouth, but he could not swallow comfortably, and wrote of that period: "I was not much improved and tried to carry on my employment in London, still attending hospital and going in overnight for dilatation frequently. Never felt free from fear because any food I took lodged in my throat; sometimes it would disperse quickly and at others it would cause severe choking and distress. The condition gradually grew worse again until in July, 1936, I was admitted to hospital three times in one month, and on August 15 remained as an in-patient at St. Mary Abbotts, being dilated frequently until transferred to Hammersmith."

For the last three months the patient noticed that food seemed to stick below the manubrium sterni. It seemed to fill up the gullet, after which he could take no more for several hours. Some pain in the chest was also noted at this time. He was not able to regurgitate

the food swallowed. On two occasions he felt something swallowed stick in the gullet and cause complete obstruction. Dilatation was no longer sufficient to allow semi-solid foods to pass. On admission to Hammersmith he could only swallow thin fluids, such as milk, cocoa, ovaltine, etc. The appetite was always good, but he felt that he could never get enough food down and hunger often kept him awake at night.

On admission, he was a spare, nervous, introspective type of individual, weighing 10 st. 4 lb. and of good colour and fair nutrition. A superficial transverse scar was visible on the anterior surface of the neck where he had attempted to cut his throat. He was only able to take liquid food and with a good deal of difficulty. The patient did not settle well in the ward and was a little troublesome. On August 28 the following report on the X-ray examination was received: "Œsophagus: The thinest medium passed down the whole length with a momentary hold-up at the lower edge of the aortic arch. With a slightly thicker bolus this obstruction became more obvious. There is a stricture at the level of the lower border of aortic arch which is permeable. There is a small amount of dilatation proximal to this. Patient is an air swallower."

On September 2 an œsophagoscopic examination was carried out, and confirmed the presence of a centrally situated fibrous stricture 25 cm. from the teeth. A plum-stone was impacted at the strictured area; this had not been noted on the X-ray films, though it was quite obvious when the films were re-examined in the light of the disclosure of the œsophagoscope. The œsophagus above this point was a little dilated, but not grossly scarred. It was possible to dilate the stricture up to No. 30. The patient was temporarily improved and able to swallow much better, but two weeks later X rays still showed very definite narrowing. When he left hospital three weeks after the dilatation, the patient said he felt sure it was closing up again. He left hospital with instructions to come back to see me on my return from vacation. On October 5, 1936, the patient wrote a letter expressing his disappointment and apprehension as to the future. In that he said: "I have been undergoing treatment for three years now. Usually dilatation with the aid of the œsophagoscope, except for a brief period, I believe in 1934, when it was possible under an anæsthetic to pass quite a large bougie without the œsophagoscope. The condition is now definitely worse than twelve months ago, yet during this period it must have been stretched no less than twenty times! If you go by my general condition, that is due to real care and good feeding by loved ones. I could, if certified fit, return to an exceptionally good situation. I am prevented from this by the fact that on several occasions I have been discharged with knowledge of 'great improvement' and as 'fit', only to find myself very soon in difficulties again, with the necessity to return to hospital. Consequently, my employers now say they will not accept my certificates of fitness unless their doctor, or inspecting doctor, is perfectly satisfied as to there being some measure of permanency in any improved condition of my œsophagus. You see my dilemma".

"You will see that there are many things which have made me want to write to you to ask that if anything can be done better than this continual wearing and not very effectual dilatation, you will do it for me. I am due to attend your clinic on Friday, October 9, and would so much appreciate something really definite. My ability to swallow is at present very poor. I have been holding out and have purposely not been again to the throat department for dilatation until I have seen you."

Two days later the patient was readmitted for reconsideration of the problem, but was very despondent when it was suggested that dilatation should again be tried. If he had been told that it was intended to make a new œsophagus or do anything equally drastic, he would have agreed without a murmur, but he very much demurred to a further trial of what he had come to look upon as an uncertain and disappointing method of treatment. As already related, he eventually agreed to my suggested plan of self-dilatation and expressed himself as very willing to co-operate. My then House Surgeon, Dr. Nicholson, and the Sister of the Ward both entered whole-heartedly in co-operation and did everything possible to help the patient. The method having been explained to him in full detail, he was given a set of small bougies and was encouraged to learn to pass them. Any lack of confidence was supplied by Dr. Nicholson, and at the first sitting the patient was able to pass a very small bougie, about quarter little finger-size, without much trouble. After that he devoted himself to the bougies about twice each day and in consequence immediately commenced to improve. The treatment was started on Oct. 13, 1936, and was continued without interruption until Nov. 10,

when the patient was allowed to return home with bougie No. 22 for regular use. He had gained 4 lb. 5 oz. in weight.

By Nov. 25 he was passing bougie No. 24 each day and taking ordinary food, and was out to a dinner party the previous night, just six weeks since he began to treat his own stricture by intermittent dilatation. On December 5, 1936, the patient wrote: "I rejoice to say that my health is marvellous; I continue to increase in weight and can now eat almost anything."

On March 5, 1937, an X-ray examination showed: "Barium paste passed down the œsophagus without delay or difficulty. No obstruction seen."

Since this date he has taken a post in South Africa, and is getting on very well, and in August of 1937 he wrote and reported continued progress. In April, 1938, he again reported. On the whole he is very well, is making long treks, playing games actively, and taking most meals with comfort. He has gained 9 lb. in weight, and judging from his photograph looks excellently well. He still uses his bougie (No. 24) about once a week. Just lately he has observed an occasional hesitation in swallowing, with a feeling as though there was something hard in his throat; he is going to use the bougie more frequently.

But there are cases in which the œsophagus is to all intents and purposes obliterated and no method of treatment by the mouth will enable the surgeon to negotiate the stricture. The case about to be reported was of that nature, and though the record gives the details, the clinical picture would not be complete if I failed to point out that this patient had the advantage of expert advice from an early stage and that every attempt had been made to overcome the disability by what I have come to look upon as the standard methods now employed.

Obliteration of Œsophagus as Result of Swallowed Corrosive—Retrograde Dilatation from Gastrotomy followed by Self-dilatation by Mouth—Restoration of Normal Swallowing.—The patient was an unmarried woman of 34 admitted to Hammersmith Hospital in August, 1935. About sixteen months previously, that is, in April, 1934, she accidentally swallowed a quantity of hair lotion instead of a tonic which she was taking at the time. The lotion was not analysed, but was supposed to contain strong ammonia and peroxide of hydrogen. She was admitted to a hospital in the North of England, where she was treated for six weeks, and on discharge could swallow foods like milk pudding. A few weeks afterwards the patient started having increasing difficulty with swallowing and the "throat felt getting tight." She had to return to hospital, where gastrostomy was performed and œsophageal bougies passed, but with only slight benefit.

When admitted to Hammersmith she could not even swallow her saliva. She had been having 4-hourly feeds and had kept fairly well.

For the previous twelve months this patient had been under the care of an expert in œsophagoscopic examination, and on June 24, 1934, this had shown that the pharynx and the upper two-thirds of the œsophagus were normal, but below this the mucous membrane was pink and velvety and there were œdematous granulations which bled easily. The condition was one of subacute œsophagitis following the action of the corrosive. At that time the patient could only swallow fluids with considerable difficulty and the dysphagia was increasing, so that she had to depend almost entirely on gastrostomy feeding. On September 19, the œsophagoscope disclosed an ulcerated area 13 in. from the teeth. The patient could still swallow fluids. Repeated attempts were made to recover a swallowed silk thread from the gastrostomy in the hope of thereby introducing a Tucker's retrograde bougie. On October 27 a barium examination showed a stricture at the level of the 10th thoracic vertebra. On November 23 œsophagoscopy disclosed a stricture 12 in. from the teeth, which narrowed to a much firmer closure 2 in. lower down. These strictures were dilated with gum-elastic bougies under the guidance of the eye. After this the patient was, unfortunately, lost sight of until May, 1935.

By that time she was in fair physical condition, but complained of much retrosternal discomfort and regurgitation of foul viscid fluid. Œsophagoscopy revealed much dilatation above the stricture and, in spite of active reflex vomiting movements, there was no regurgitation of stomach contents. The stricture was seen at 15 in., and there was a great deal of

scarring of the mucous membrane for some 3 in. above that point. Only a fine gum-elastic bougie could be passed through the narrowing. The dilatation was repeated a few days afterwards and was accompanied by slight hæmorrhage. On June 3 some fresh granulations were noted just above the stricture, and during the dilatation it was thought that a false passage had been created. After this the condition got much worse and there appeared to be total obstruction.

At the time of admission to Hammersmith Hospital, the patient was found to be in fair general condition. Red blood-count 4,500,000, hæmoglobin 84 per cent. Well nourished, of apparently healthy mentality, and weighing 7 st. 4 lb. While speaking she did not appear to be troubled with accumulation of saliva, but the sputum jar by her bedside was found to contain about 10 oz. every 12 hours. Nothing abnormal was discovered on examination of the chest or abdomen.

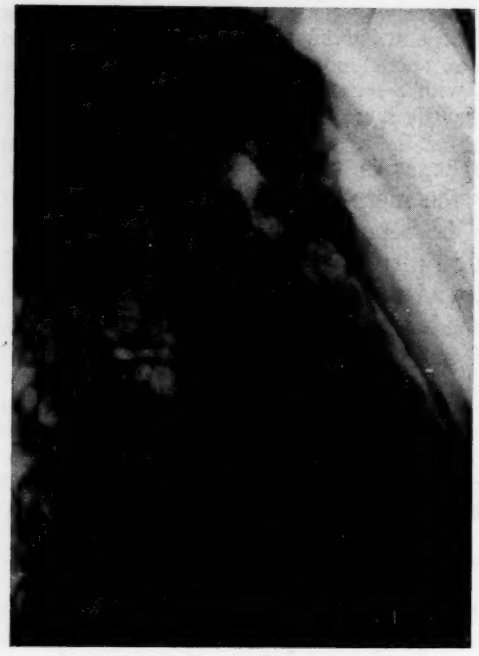


FIG. 392.—Cicatricial stenosis resulting from swallowing of hair dye.

An X-ray examination made on August 10 showed what was regarded as an obliteration commencing about an inch above the lower end of the œsophagus, which tapered down to this point. No opaque material could be demonstrated passing through. (*Fig.* 392.) With the assistance and willing co-operation of the patient, attempts were made to pass bougies, but the smallest instruments could not be got through. A ureteric bougie passed down to 15 in. from the incisor teeth, but X rays showed that the instrument was coiled up in the dilated œsophagus above the obstruction. Further attempts were unavailing, and it was quite clear that some other plan of treatment had to be devised, and especially as the Plummer silk-thread method had entirely failed. It was, therefore, decided to explore the lower end of the œsophagus from the abdomen in the hope that it might be possible to mobilize it sufficiently and to bring it down so that an operation of the type of cardioplasty could be carried out. If this was not possible, retrograde dilatation appeared to be the next step rather than the contemplation of the formation of a new œsophagus.

OPERATION.—On September 17, under general anæsthesia, a left paramedian incision was made. The gastrostomy opening was plugged and excised. The peritoneum was opened around the gastrostomy and the stomach delivered, a curved clamp being applied to control the gastrostomy opening. The lower end of the œsophagus was brought to view by mobilizing the left lobe of the liver after dividing the left lateral ligament and retracting the spleen. At the lower end of the œsophagus, commencing about an inch above the cardia, was a hard firm mass, about the size and shape of the thumb and about $2\frac{1}{2}$ in. in extent. There was considerable inflammatory thickening and fixation round about and the affected part of the œsophagus could not be drawn down into the abdomen, so that a local plastic operation was out of the question. An opening was therefore made into the stomach near the cardia and a No. 6 bougie introduced and guided into the stricture (*Fig.* 393). With a little steady pressure it went through and was advanced until it appeared in the mouth. A $\frac{3}{8}$-in. soft rubber tube was then slipped over the upper end of the bougie and drawn down through the stricture into the stomach where the lower end was stitched to the gastric wall with catgut. The opening in the cardiac end of the stomach was then carefully closed in layers. The old gastrostomy opening was utilized for the implantation of a fresh gastrostomy tube by Senn's method. The abdominal incision was then closed. The patient stood the operation perfectly well and it appeared as though the prospects were good.

POST-OPERATIVE PROGRESS.—She made a good immediate recovery, but three days afterwards she looked rather ill and complained bitterly of the tube, which was therefore removed

from the mouth. This required a good steady pull. Directly afterwards the patient flushed in the face, looked very ill, and was considerably upset and complained of pain in the epigastrium and in the chest. Next day the patient was seen by Dr. Scadding, who came to the conclusion that she had some slight collapse of the right base. This condition quickly improved.

Ten days after operation the patient was assisted to use œsophageal bougies, commencing with No. 6. She managed quite well and the treatment was continued daily, the sizes being gradually increased. By Oct. 6 she was able to pass Nos. 13 and 14 bougies fairly easily. The general condition was greatly improved and the patient took very little food by the gastrostomy, as she was able to swallow so much better. She was gaining weight.

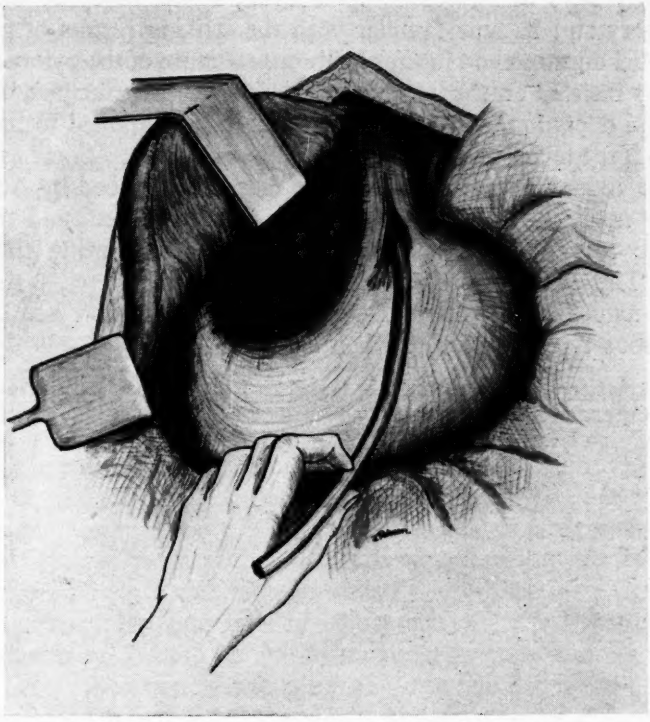


FIG. 393.—Retrograde passage of bougie. Note the opening in the stomach wall very near the cardia.

On Oct. 18 the gastrostomy tube was removed and X-ray examination showed that an opaque meal of the consistency of cream passed without difficulty through the stricture, the lumen of which was much larger than when first examined. Opaque material of the consistency of porridge also passed, but this thicker substance demonstrated partial stenosis, with proximal dilatation. Twenty minutes after the administration of the meal, the œsophagus was completely empty.

The patient eventually made a good recovery and was able to leave the Hospital on Nov. 13. She was then passing a bougie No. 18, and this she took home.

At the end of February, 1936, the patient wrote, "Please allow me to thank you for the perfect health which I now enjoy". She was using No. 19 bougie and taking food very well. She stated that if she neglected to chew her food thoroughly, there was a momentary sensation of narrowing at the bottom of her throat, but that otherwise she had no symptoms whatever.

A year after leaving the hospital it was reported that she was in excellent health. She had no trouble of any sort and had not used the bougie for the previous four months and had gained 2 st. 7 lb. since the operation. At this time X-ray examination showed no evidence of obstruction.

In March, 1938—that is, 2 years and 4 months after leaving hospital—this patient wrote as follows: "It gives me great pleasure to inform you that I'm now enjoying the best of health. I have only had the occasion to use the bougie twice in the last year. I take ordinary food quite the same as the rest of the family without any difficulty. My present weight is 9 st. 2 lb., just as it was before my illness."

The experience of this and other cases taught me that if it is desired to attempt to pass bougies by the retrograde method it is necessary to make the opening in the stomach as near to the cardia as ever possible, despite the fact that in this situation the incision in the stomach is rather difficult to suture.

In my early days in Newcastle-upon-Tyne, I saw much of the development of gastric surgery and became familiar with the striking results of pyloroplasty by the Heineke-Mickulicz method in simple fibrous stricture of the pylorus. Rutherford Morison always insisted on the general application of the principles involved in that really wonderful operation, and I knew that it might be applied to the lower end of the œsophagus if the technical difficulties could be overcome. Some experience in the exposure of the abdominal œsophagus by the method described by Adrien Lambert, of New York,[3] made me familiar with the problems involved, and therefore, when a suitable case turned up, I had no hesitation in attempting the operation of cardioplasty. The brief record of the following case presents the main facts. Whatever the nature of the original pathology, the condition had developed into the simple fibrous type of stricture when I operated.

Fibrous Stricture of Congenital Origin—Failure of Dilatation—Recurrence after Heller's Operation—Cardioplasty followed by Cure.—The patient (Fulham) was healthy in infancy and early childhood, but at the age of 8 years and 6 months began to

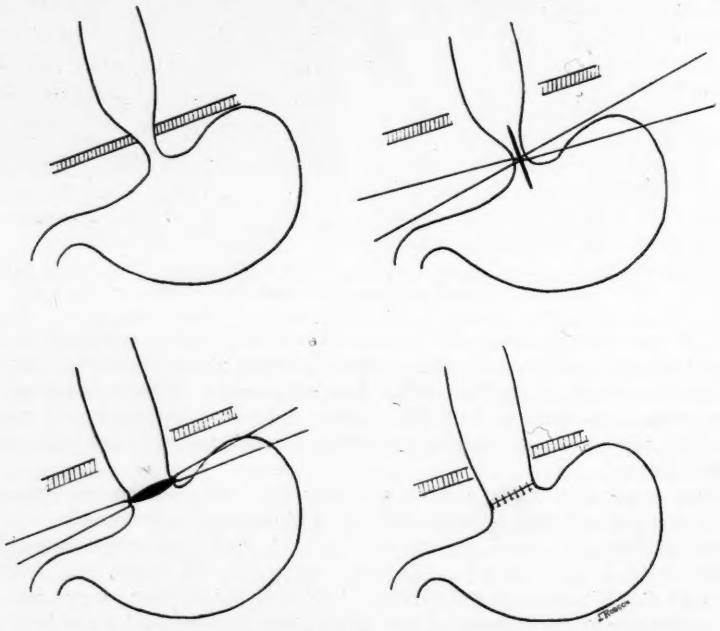


FIG. 394.—Diagrammatic representation of the operation of cardioplasty.

"vomit" immediately after taking food. There was no pain, but he rapidly wasted and his condition caused much alarm. At this stage he was treated by physicians, and was kept in hospital on more than one occasion and for various periods, but with only temporary benefit.

In August, 1924, when the boy was just over 10, one of my colleagues exposed the lower end of the œsophagus and carried out Heller's (1913) operation. The patient made an easy recovery and was much better during the few weeks he remained in hospital, being able to take light food by the mouth, but as soon as he returned to his home he began to "vomit" as before, and steadily lost ground.

By this time both the patient and his parents were naturally very disappointed, and the boy would not return to the hospital. Finally the condition became desperate; he was scarcely able to swallow at all, he had lost weight to a serious degree, and had become intensely anæmic and generally œdematous. In this state he was finally re-admitted to hospital in March, 1926, being then 12½ years old. His condition was very alarming and I have never seen anybody so starved. After a short preparation to overcome dehydration, I carried out gastrostomy, using local anæsthesia. The boy rapidly improved, and in two months it was considered that he was well enough to justify a further operation on the œsophagus. This time a definite cicatricial stenosis of the lower end was disclosed, and this was treated by cardioplasty (*Fig.* 394). The vertical incision was 1½ in. long and was carried into the lumen throughout its length. Recovery was rapid and complete, and almost from the first he was able to swallow without difficulty. This patient has been under close observation ever since the operation. In May, 1931, he was well and strong and working regularly, and wrote, "I take normal food like a normal person". He has continued in good health and at the present time (1938), now twelve years after operation, he is a tall, well-developed, young man, able to work and play like his fellows and to take his food without the slightest hesitation. There has never been any trouble with swallowing since the operation and he has never required further treatment, nor has a bougie ever been passed.

In another somewhat similar case in a man aged 50, but owning a different aetiology, the result has been equally satisfactory, and now five and a half years after operation he continues to enjoy good health and swallows normally.

On the occasion of that most productive Vienna visit of which I have already spoken with such enthusiasm, I saw much of the work of Professor Hochenegg in the surgery of malignant disease of the rectum, and I have never forgotten the extremely valuable lessons which I then learnt. But the activities of his clinic were not limited to any one branch of surgery, and I was struck by the story of a case of œsophageal stricture to which he casually referred as we went through his wards. It was that of a young man in whom one of his assistants had made an anastomosis between the dilated œsophagus above a stricture and the cardiac end of the stomach. This operation was carried out through the chest and the patient had made a very good recovery. This always appeared to me a surprising surgical exploit, and I longed for an opportunity to try it, but I had to wait until after the world war, an occasion presenting itself in 1919.

Fibrous Stricture following Achalasia—Complete Failure of many forms of Treatment—Permanent Relief after Transthoracic Œsophago-gastrostomy.—A young man of 19 came under my observation, complaining of increasing difficulty in swallowing for two and a half years. All kinds of treatment had been meted out, with little success, and at one period he had become so ill that gastrostomy was necessary to ward off impending starvation. After that, bougie treatment of various sorts was again carried out, but with only incomplete and temporary relief. After some further time the poor lad came to me in great distress, begging for something to be done. The gastrostomy had been allowed to close, he could scarcely swallow at all, and had become an outcast, as he was looked upon as a nuisance by his family, who grudged him suitable nourishment since he was too feeble to work. With such a free hand, I had no hesitation in carrying out the operation with which my Vienna visit had inspired me. It was difficult and trying, and convalescence was attended by much anxiety, but the lad eventually recovered and was able to swallow sufficiently to be independent of any other means. He took ordinary food with the rest of the family, returned to work, became a self-supporting citizen, and was contemplating matrimony when unhappily, just two and a half years after the operation, he was knocked down by a motor

car and received injuries which proved fatal. By the merest chance I heard of his death and was able to secure the specimen at a post-mortem. The original stricture at the lower end of the œsophagus was so small that it would only admit a very fine glass rod.

I have now carried out this operation on 5 other occasions using an abdominal approach.[4] There has been no death in the series. In one case the patient was not relieved, but the others remain well after periods of from seven to nearly fourteen years.

THE CAUSES OF NON-MALIGNANT STENOSIS

The greater number of examples of this condition are due to the swallowing of corrosive solutions (*Fig.* 395), and in these circumstances the causative factor is quite obvious. The condition may also follow the healing of traumas associated with foreign bodies or with attempts at their removal. Fibrous tissue is always associated with Nature's efforts to repair the damage resulting from inflammation, and for this reason stenosis may follow the healing of an ulcer. A peptic ulcer at the lower end of the œsophagus may heal with the development of fibrous stricture, and similarly the cicatrization of a high-lying gastric ulcer may leave a scar involving the lowest part of the œsophagus. For the same reason, an infected gland, for instance near the root of the lung, which invades the œsophagus may be the cause of a stricture. Destruction of the mucous membrane may be due to direct invasion from neighbouring parts, and, for example, an acute infection of the throat and pharynx may spread down the œsophagus, or the latter may be infected by the diphtheric process, as in cases recorded by J. D. Rolleston.[5] Simple strictures have also followed on scarlet fever, typhoid fever, the vomiting of pregnancy, and some other similar conditions. I have often wondered what the explanation may be in these latter cases. I was much impressed with the work of Pringle Stewart and Teacher[6] on the digestion of the œsophagus, and though I have not seen any of the extreme instances of this condition, such as they have recorded, I have frequently seen lesser degrees in patients dying from a variety of conditions. Professor J. Burton Cleland, of Adelaide, in a thousand post-mortem examinations in which he systematically examined the œsophagus, found occasional erosions, local necroses, and ulcerations in patients dying from general diseases.

FIG. 395.—Stricture of œsophagus the result of swallowing hydrochloric acid nine months previously. Patient, a woman of 38, lost 6 st. in six months.

It seems to me highly likely that in many of the conditions mentioned patients who are extremely ill may regurgitate acid and possibly toxic stomach contents into the œsophagus, with erosion as a result. Quite likely in most cases as the patient recovers the œsophageal erosion clears up, but it is not improbable that in

some it does not do so entirely, and the condition only slowly gets well at the expense of the development of fibrous tissue leading to stricture.

It has for long been suggested that syphilis may be the cause of fibrous stricture of the œsophagus. I cannot deny that this may be so, but at the same time I have never been able to satisfy myself of the syphilitic nature of any lesion of the œsophagus which I have either seen clinically or as a pathological specimen (*Fig.* 396). I am therefore forced to the conclusion that this disease very rarely attacks the œsophagus, and that it is a mistake to consider it among the probabilities.

FIG. 396.—Narrowing of the œsophagus, with leukoplakia and simple inflammation. The patient, a woman of 54, gave a twelve months' history. The condition may have been luetic in origin.

But even if we bear in mind all the conditions which I have mentioned as possible causes of non-malignant stricture, there are still a considerable number which cannot be accounted for in any of these ways. Some of them are probably a relic of a congenital narrowing to which has been superadded some sclerosing process at a later period in life. Brown Kelly, of Glasgow,[7] has produced some convincing evidence to show that such a sequence of events is not unusual, and one must admit that it is a likely explanation in many of the cases. For instance, in the series of 19 examples of non-malignant stricture of the œsophagus referred to in this paper no less than 5 probably own a congenital ancestry. Although cardiospasm or achalasia is not usually associated with gross structural change in the wall of the œsophagus, this condition may be a direct cause of such a change in a few of the cases. In the first instance, it is probably simply a neuromuscular defect, but as a consequence dilatation of the œsophagus occurs and there is some resultant stasis. In some cases this stasis in turn leads to inflammatory changes in the mucous membrane, which may extend to the deeper coats, and once that has occurred the development of fibrous tissue and subsequent organic stenosis may readily follow. In my series that was probably the explanation in four cases. But it is a mistake to assume that these changes occur in all cases of long-standing achalasia, or indeed in many, and I have no information to show at what stage of the disease they may be expected to develop.

I have very definite evidence of what I suppose is common knowledge, that the condition of achalasia may remain as a neuromuscular defect for many years. In a recent case, a woman of 34 came under my care suffering from an œsophageal obstruction for which gastrostomy had been carried out eleven and a half years previously. This patient assured me that during those years she had never been able to swallow anything more than a small cup of tea, and very often that was ejected. The appearances were characteristic of achalasia, but under observation for twenty-five minutes none of the opaque material was seen to find its way into the stomach. In these circumstances I felt sure that we were dealing with an

example of secondary cicatricial stenosis developing on achalasia, but, nevertheless, I felt that one ought to try the effect of a mercury-filled bougie. To my great astonishment the latter slipped through the obstruction into the stomach without the slightest hesitation, and the patient was able to pass such a bougie herself without difficulty. As a result, the power of normal swallowing was restored, until death from an acute pulmonary infection carried her off some months later. Unfortunately it was not possible to secure a post-mortem examination.

In the 19 cases referred to in this paper, the causation, so far as it could be ascertained, was as follows: corrosives 5, congenital 5, late results of achalasia 4, following ulceration 2, acute inflammation 1, undetermined 2.

The usual sites for simple strictures of the œsophagus are well recognized as being at the junction of the pharynx with the œsophagus, at about the crossing of the left bronchus, and at the termination of the œsophagus. But such a stenosis may extend along the œsophagus for a considerable distance, or there may be multiple strictures. In form the stricture may be annular, or it may be irregular, and the orifice may be central or eccentric. I would like to suggest that probably the anatomy of such strictures when in situ in the living body is not quite the same as when the part is available for examination after post-mortem removal.

SYMPTOMATOLOGY

This is usually a question of gradually increasing dysphagia, until such time as the patient develops extreme difficulty in swallowing even water. Sooner or later, if the condition is unrelieved, the day arrives when even saliva has either to be ejected or to accumulate in the œsophagus, only to be regurgitated at intervals. In many of the cases the patient gives a history that at times the condition is much worse then others, and it has been usual to assume that this is explained by a superadded spasm. It is more likely that such variations are due to the temporary blocking of the opening by the lodgement of food particles or by temporary swelling of the mucous membrane in the neighbourhood as the result of infection. In the definitely traumatic cases, the interval between the infliction of the injury and the development of the stenosis varies a good deal, and whereas the latter may develop perhaps as soon as six weeks after the injury, it may be deferred for months. Nevertheless, it may safely be said that, once set up, the stenosing process is continuous and that the tendency is for it to become steadily worse.

DIAGNOSIS

Though experience has taught me that it may be very difficult to be sure of the exact aetiological factor in some of these cases, it is usually at least easy to say that the condition is non-malignant. But there may be difficulties, and in this connexion I always recall a case of carcinoma mistaken for simple stricture which came under my observation nearly forty years ago, before we had the advantage of the diagnostic methods which we now employ. But this error in diagnosis is not unknown even in these days, and during the last two years I had a patient under my care in whom it occurred.

The patient was a woman, 54 years of age, who came to the Hammersmith Hospital on July 7, 1936, complaining of difficulty in swallowing of three months' duration. She had been able to swallow fluids without difficulty and did not think that the trouble had been

getting much worse since the onset, though she had lost some weight. In the middle of June she went into another hospital, where a diagnosis of carcinoma of the œsophagus was made.

On admission under my care she was found to be a healthy looking woman, who worked as a cleaner and had been accustomed to eat her meals rather hurriedly. There were no general signs of disease of any sort, but radiography disclosed an obstructive lesion 3 in. above the diaphragm, but without any filling defect. The proximal part of the œsophagus was dilated. Even the thinnest opaque medium was held up, though the stricture was not absolutely impermeable. Its outline was perfectly smooth and the channel through the obstruction was strictly central in position (*Fig.* 397). The X-ray appearances therefore suggested a benign stricture, but it was remarked that an annular malignant stricture could not be excluded. An œsophagoscopic examination was made shortly afterwards and disclosed a perfectly smooth, fibrous-looking narrowing without ulceration or tendency to bleed, The condition found was looked upon as a simple stenosis without any suggestion of malignancy. A good-sized œsophageal bougie could be readily passed through the constriction. No biopsy was made.

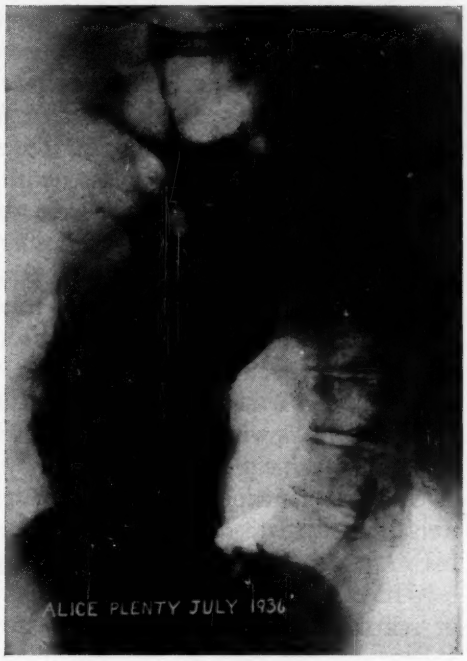


FIG. 397.—Malignant stenosis which was mistaken for a simple condition and treated by dilatation.

In view of the diagnosis of non-malignant stenosis, treatment by Hurst's loaded bougie was commenced. Size No. 18 was held up for a moment at 12 in. from the teeth and then slipped on easily into the stomach. The bougie was used daily, and by July 22 a No. 20 could be passed without difficulty. On leaving hospital she was provided with a mercury-filled bougie, No. 22, and this she continued to use regularly for eight months. During all this time she was very well, was taking ordinary food, and was able to continue her work, but she did notice that there was a steady loss of weight.

She was re-admitted to the hospital on April 28, 1937, when she informed us that there had been a considerable amount of hæmorrhage after passing the bougie a fortnight previously, and from that time onwards the instrument would not go down easily. She had tried passing the bougie on alternate days, but every now and again its use was followed by bleeding. During this period there had been considerable difficulty in swallowing, and she now felt great discomfort and there was regurgitation of solid food.

On examination the patient was found to be very emaciated, weighing only 5 st. There were quite obviously secondary deposits in the liver. On May 5 the abdomen was opened. Not only were there masses in the liver, but there was a large lump in the lesser omentum just beyond the cardiac opening. Gastrostomy was made in the usual way and this gave the patient great relief, and she was able to leave hospital on May 29, but in August she was released by death from the pangs which art had only temporarily relieved.

THE MANAGEMENT OF SIMPLE STRICTURE

I believe it was Chevalier Jackson who first advised that all treatment ought to be based on œsophagoscopic examination of the local condition. He taught that dilatation under the guidance of the eye can usually be carried out, and laid it down as a dictum that the great majority of strictures, which can be seen and negotiated by this route, can be cured by repeated and painstaking dilatation. In

his writings he points out that webs can be made to disappear after a few treatments, but that strictures involving the entire periphery of the œsophagus will require a longer time, and that those of considerable linear extent are more troublesome still. The routine plan is to dilate these strictures with tapering bougies, which are passed in succession up to as large a size as can be got through at any one sitting. Such dilatations are repeated at regular intervals, depending upon the reaction in the individual case. There can be no question that the treatment of most strictures is largely a matter of the proper use of bougies and that they ought to be the first and the main method of treatment in all cases, whether carried out under the guidance of the eye through the œsophagoscope or without its aid. But although I know that very many cases are successfully treated through the œsophagoscope, my experience has taught me that failures are not uncommon, and these are reflected in the story of the cases which I have related. But bouginage without the guidance of the eye should be by the method which I learned at the hands of Professor Lotheissen, for it is essential that the stricture should be gently dilated and that traumatism from over-stretching or from tearing should always be avoided. The problem is analogous to that with which the surgeon is confronted in dealing with urethral stricture, and I have the most unhappy memory of the old days, when, as a dresser and as a junior surgeon, one used to over-dilate such strictures, taking pride in the large size of the bougies which could be forced through. I now know that in most of those cases the stricture was torn rather than dilated, and that it was the process of repair associated with such traumatism that accounted for the subsequent difficulties which we so often encountered. It fills me with remorse when I think of the many occasions when, with the best possible intentions, I must have unwittingly inflicted such traumatism without realizing the violation of surgical principles involved. The older surgeons were well aware of the need of care and gentleness in dealing with urethral stricture, and the student would be well advised to read the wise remarks on this subject made by Robert Liston in his *Practical Surgery*, published as long ago as 1840.

In connection with the *treatment by bougies*, there are certain matters worthy of remark. The question of the sizes has always been rather confusing to me. I have no difficulty in understanding sizes expressed in terms of the diameter in millimetres, but I have found it difficult to carry in mind the various scales which are in use. I find it convenient, though admittedly it is not scientific, to speak about œsophageal bougies in terms of the size of the digits. An instrument equal to the size of the middle phalanx of my little finger is 14 mm. in diameter, so that a " half-little-finger size " is 7 mm., and a " quarter-little-finger size " is $3\frac{1}{2}$ mm. For bougies smaller than this we come down to instruments resembling ureteric catheters, and as a matter of fact in some of my cases I have commenced treatment by passing bougies of that small size or by actually using these catheters. The corresponding phalanx of my first finger is equal in size to a bougie of 16-mm. diameter, and that may be conveniently spoken of as a large size œsophageal bougie. I use bougies of the French variety, which I believe are manufactured largely of coated silk and are pliable instruments, especially when gently warmed. They have slightly tapering ends, but they are not of the rapidly tapering shape which are sometimes employed for use through the œsophagoscope. When the time arrives that a bougie of little-finger size can be passed, then it may perhaps be followed by the smaller of the mercury-filled bougies, associated with the name of Sir Arthur Hurst, and which are supplied

in sizes from 13 mm. to 20 mm. in diameter. For purposes of lubrication, I find that there is nothing so good as olive oil. If really good cream is available, then it may be used as being more pleasant, but it is not so efficient as oil. A convenient compromise is to get the patient to endeavour to swallow a little cream, but to lubricate the bougie with olive oil as well.

The technique of the method of *self-dilatation* consists in explaining to the patient what is to be done and insisting that it is not a question of 'thrusting' a bougie down the throat, but of endeavouring to 'swallow' the instrument, which may be assisted in its passage by gentle manipulation. No force must ever be used, nor is it ever necessary to produce the slightest pain. It is essential to explain to the patient exactly what is to be done, and in my cases I have made a point of getting the co-operation, not only of the patient, but also of some sympathetic nurse and of the house surgeon. On the first occasion, it is essential that patients should be assisted, as they may just fall short of success because they are a little afraid to use that slight amount of manipulation which may enable the bougie to engage in the stricture when reached. The patient's bed is surrounded with screens or, better still, the patient is allowed to sit alone in the day-room or some quiet place. The appropriate bougies are provided and the patient is encouraged to experiment with them to get confidence and to learn to pass the instruments unaided. All of my patients have ultimately learned to manage their own bougies, though some of them have declared at the outset that they would never be able to overcome the disinclination to let the instrument go over, and, of course, some few patients are not co-operative.

The question of how often the bougie should be passed depends on the type of stricture and on the ease with which the instrument goes through. If, for instance, at a first sitting, it is possible to pass bougies up to half-little-finger size and the patient can swallow better afterwards, then probably every second or third day will suffice for the treatments. If, on the other hand, it is only with great difficulty that an instrument can be got through, it will be essential to use such an instrument every day until there is a tendency for the resistance to give way. If the bougie can only be got through with great difficulty, or if it is gripped, then it should be left in situ for three or four hours and the process should be repeated every day for a time. In many cases it will take a week or even longer for the patient to work up to a little-finger size bougie, and perhaps a fortnight or more to get up from that to a fore-finger size. Having arrived at this size, the patient is to be encouraged to take ordinary food, so that each bolus will itself act as a dilating agent. It is probably better, even in the most favourable cases, to use the bougie perhaps twice a week for three months, and then once a week for six months, and once a fortnight for as long as two years. If after this time there appears to be no tendency to recurrence, then perhaps the bougie can be dispensed with, although most patients feel that they prefer to pass it now and again as an insurance. But if there is even the least tendency to closing up of the passage, the more frequent use of the bougie should be resorted to without the slightest delay and for as long as such a tendency exists.

The *prevention of stenosis* in the presence of such conditions as are almost invariably followed by its development is an important aspect of the matter. There is a school associated with the name of Salzer who advise the passage of instruments in the very early days, although it is traditional to believe that it is better to defer such treatment until several weeks after the accident. In the following case, perforation

of the œsophagus was caused by a bougie passed on the twenty-sixth day after the swallowing of nitric acid.

Injury of Œsophagus from Swallowing Nitric Acid—Perforation by Bougie—Acute Infection and Death.—The patient was a young woman, aged 22, who came under my observation early in 1906. In a fit of depression she swallowed about 2 oz. of nitric acid on December 23. An emetic was given and she was apparently getting on very well until January 19, 1907, when she began to have some difficulty in swallowing; up till then she had been able to eat meat and potatoes. At that time she seemed in good health, except that she spat up some mucopus. A full-sized œsophageal bougie was passed with ease; there was no pain, but a little blood was noticed on the bougie. Just afterwards the patient took a drink and complained of pain, but she continued to swallow milk and eggs. The next day she was admitted to a private hospital very ill, collapsed, cyanosed, and with a small feeble pulse of 140 and a temperature of 102°. That evening she was found to have surgical emphysema at the root of the neck and pneumothorax on the right side. The patient died about twenty-eight hours after the use of the bougie, the friends dating the fatal illness to the instrument.

At a post-mortem examination the right pleural cavity was found full of stinking gas and contained 3 pints of fluid like curdled milk and of acid reaction. The pleura was covered with lymph and the lung completely collapsed. The heart was pushed to the left side. There was gross emphysema limited to the mediastinum and the root of the neck. As there was no hole in the lung, the pneumothorax was probably due to swallowed air passing directly through the perforation, but it may have resulted from decomposition in the pleura. The upper half of the œsophagus showed the result of sloughing, and there was much granulation tissue, with a narrowing about the middle. The bougie which had been used would not go through this narrowed area. Behind the upper half of the œsophagus there was an abscess, and from this an aperture led through its wall into the cavity of the chest. The pleura at the perforated spot was sloughing, as if the abscess was on the point of bursting when the bougie was used. A bougie passed from the œsophagus went easily through the rent into the pleural cavity. The left lung was healthy. The mucous membrane of the stomach showed considerable destruction.

I should hesitate very much to attempt to pass any instrument until the stage at which fibrosis has superseded the active inflammatory changes resulting from this sort of trauma. This will probably seldom be less than six weeks, and may be much longer. Difficulty in swallowing at an earlier stage, and especially if associated with pain and the expectoration of mucopus tinged with blood, is probably an indication for temporary gastrostomy. Quite possibly an earlier resort to the production of physiological rest by this means would limit the inflammatory trouble and render subsequent stenosis less likely.

But there are cases in which it is not possible to pass bougies, and in these one is glad to be able to fall back on the use of the swallowed thread as a guide, a method which was first brought to my own notice in the Mayo Clinic. This method dates back a long way in the history of the surgery of the œsophagus. In the first instance, von Hacker and Dunham allowed a swallowed thread to find its way out of a gastrostomy and on to this they threaded bougies, which were guided through the stricture from below upwards. It was Mixter in 1909 who recognized the value of the silk thread without the gastrostomy. This method was further developed by Plummer in the Mayo Clinic in 1910. At that time he had used it in over 300 cases, of which 8 were of cicatricial stenosis, and there was only 1 in which the thread failed to pass through the stricture. Later on, Vinson took up the method, and it has been used by many others and with considerable success. But the method sometimes fails, and there are certain troubles associated with it which have to be borne in mind. If neither bougies nor the silk thread can be persuaded to negotiate the narrowing, then

it is suggestive that obliteration is almost complete, but even when this occurs it is quite possible that retrograde dilatation may still be successful. In my experience it is not easy to guide an œsophageal bougie into the cardiac orifice from a gastrostomy and I know that failures are not uncommon. Even with the stomach freely exposed it may be difficult to get a bougie to enter the œsophagus unless the opening in the stomach wall is made near the cardiac orifice, as shown in *Fig.* 393. As alternative methods, division by Abbé's string saw or by diathermy have both been recommended, but it is difficult to determine the length of the strictured area or to determine the depth of the necessary division. As a result of these methods bronchial fistulæ have been produced and deaths from perforation have occurred.

The Dangers of Bouginage.—I think it was Trousseau, the French clinician, who said that a patient using bougies would sooner or later die of the instruments, and, of course, we must recognize that there is danger in this treatment. Nevertheless, such experience as I have would lead me to conclude that the careful self-passage of bougies, blind though it may be, is certainly not attended with greater danger than the more rapid dilatation carried out under the guidance of the eye through the œsophagoscope. But laceration and perforation have both occurred, though I believe that there is often some pathological condition associated with the stenosis which softens the œsophageal wall and renders perforation more likely to occur. In a comparatively recent case which came under my notice, death followed laceration of the œsophagus by a bougie, but post-mortem examination showed that the presence of a peri-œsophageal abscess was a predisposing cause. Nevertheless, the possibility of such an accident must be ever in mind. Force is never permissible, and it should be an absolute rule never to pass a bougie blindly under general anæsthesia.

The Fate of the Stenosis Treated by Dilatation.—It was the late Professor John Chiene, of Edinburgh (1843–1923), who was accustomed to say in dealing with urethral stricture that " once a stricture, always a stricture ", and the recollection of that aphorism reminds me to speak of the ultimate condition of patients who have been treated for cicatricial stenosis of the œsophagus. One can well believe that many patients, possibly the majority, may be permanently cured by persistent treatment, but I think we must realize that there is probably a residue of cases in which the tendency to recurrence persists for many years and possibly throughout life. One of my patients, who first showed signs of such a lesion at the age of 3, was later treated by dilatation with a satisfactory result. Now at the age of 16 she is able to swallow normal food like the rest of the family, but having of late neglected the regular use of the bougie, she gets some pain behind the sternum, and a radiograph shows that there is a well-defined, though temporary, hold-up at the lower end of the œsophagus. The record which follows gives the details of what has proved a very interesting case.

Congenital Stenosis under Observation for Fifteen Years—Gastrostomy—Spontaneous Restoration of Swallowing—Recurrence Successfully Treated by Self-dilatation—Relapse after Four Years.—This child (N. S.) was just under 3 years of age when she first came under my observation in 1923, with a history of " vomiting " from birth. This was much worse at times, when the child could retain so little food that the parents gave up hope of being able to rear her. The girl was very small and thin and very much under-nourished, weighing only 10 lb. at ten months. X-ray examination disclosed stenosis near the lower end of the œsophagus. Unfortunately, an endoscopic examination

was not carried out. As bougies could not be passed, the abdomen was opened and the lower end of the œsophagus explored. A constriction was felt just above the diaphragm, but a bougie could not be passed through it from the mouth, nor could one be directed into it from below. Gastrostomy was carried out. About four days later the child collapsed and nearly died, but soon picked up again. At this time the parents were not co-operative and would not permit further attempts at treatment. At the end of a month the gastrostomy tube was removed and the child was taken home.

After leaving hospital she was able to swallow liquids on most days, but not every day, and nearly always regurgitated some of the food taken. The child continued thin, small, and weedy, but when 6½ years old, and without any obvious reason, she suddenly began to swallow with comparative ease and was soon able to take ordinary food. When seen in January of 1931, the child was 8 years old, was very well, and weighed 4 st. 4 lb.

In August, 1932, although the child was still fit and well, she was said to get an occasional attack of œsophageal spasm. In these attacks she sometimes regurgitated food or by an effort just managed to keep it down. These attacks were very infrequent, and as a rule she could eat anything without trouble. Shortly afterwards the difficulty in swallowing became progressively worse. On July 4, 1934, her mother wrote explaining about the child's difficulty and asking that arrangements might be made to see her. Before this could be carried out, the child was admitted as an emergency on July 9 with the story that two days previously she had accidentally swallowed a cherry stone, which stuck in her throat, since when she had not been able to swallow anything.

On re-admission the child was thin, ill, and in a state of very poor nutrition. For these reasons, it was considered that the element of starvation ought to be dealt with at once, and therefore it was my intention to make a gastrostomy in the first instance and later to investigate the local condition. It was during the administration of a general anæsthetic that the child retched and brought up the cherry stone. In view of this event no further interference was considered necessary at that time, and it was found that the child could swallow fluids reasonably well. After a day or two she picked up so much that an X-ray examination could be made, and this showed a long, smooth stricture at the lower end of the œsophagus, with only a moderate amount of dilatation above it. The child was therefore taught to pass bougies and soon became quite expert. A little over a fortnight from the commencement of this treatment she was swallowing very well, and was able to take ordinary full diet. The child passed bougies daily, and left hospital on July 30, taking a series of bougies home with her. By October 2 the patient could eat any type of food and there had been no trouble since leaving hospital.

In February, 1936, the patient was in splendid health, was well developed, and was leading a normal life and had not required to pass a bougie for six months. She did not have any anxiety over her diet and literally ate anything that was going.

In March, 1938, this patient contined very well and able to take ordinary food, but had at times persistent high epigastric pain and occasional regurgitation. This pain usually came on at night and was often relieved by eating a dry biscuit. A further radiograph showed a dilated œsophagus with a marked narrowing of its lower third. For the last two years she had been so well that the bougies had been neglected. Nevertheless, she found she could pass her full-sized bougie, but it was gripped firmly and reproduced the pain. The patient is now again under treatment.

In commenting on this case, I cannot help feeling that if the dilatation had been continued regularly, the stenosis might ultimately have been overcome. But as long as dilatation has to be continued we cannot speak of cure, and until the time arrives when patients can go on without treatment for years and without the slightest trouble, cure has not been obtained, though palliation may be entirely satisfactory.

If the case just recorded was still under my care I would propose a further course of thorough dilatation, with the regular use of a bougie at increasing intervals over a period of years. If this was not satisfactory I believe that cardioplasty would enable one to deal adequately with the lesion, and that complete and lasting relief would result.

The Value of Gastrostomy as a Stage in the Treatment of Non-malignant Stenosis.—By this operation the surgeon applies the principle of physiological rest, and the consequences are often very striking. It is not only that the feeding difficulty disappears, with consequent mental relief, but the rest to the site of the stricture allows inflammatory mischief, with its attendant swelling and congestion, to subside. As a result there is often an improvement in the swallowing capacity, and the patient can more readily use bougies. The fact that the means of feeding is assured allows time for detailed investigation and treatment by other methods, while the inconveniences of gastrostomy feeding encourage the patient to persevere with the method of dilatation in the hope of restoration of the normal swallowing capacity. Gastrostomy is also an essential preliminary, or at least a wise accompaniment, of plastic operations involving incisions into the lumen of the œsophagus. I can never understand why gastrostomy is so often looked upon with disfavour, for I consider it one of the most valuable operations in surgery. But if it is to be carried out, it should not be delayed until the patient is at death's door from starvation. It is also essential that patients with gastrostomy should be adequately fed on a properly balanced diet, and that that the psychological factors, which are such aids to nutrition, should not be neglected.

Direct Surgical Attack on the Strictured Area.—With all the aids we can employ, there are some cases that cannot be treated by dilatation, and the question of direct attack on the stricture arises. Those who have no actual experience of the surgery of the œsophagus may naturally think that excision of the strictured area with end-to-end anastomosis presents a ready solution. But the human œsophagus does not lend itself to this method ; there is too great longitudinal tension as well as many other difficulties, and the plan has almost invariably proved fatal. Some few cases of stricture at the lower end may be successfully treated by cardioplasty or by anastomosis, and I have recorded several examples. But there are cases that have defeated the various methods I have mentioned because of the anatomical situation of the lesion or its extent, and for this group there is always the possibility of short-circuiting the disability by making a new ante-thoracic œsophagus, and to this end a vast amount of ingenuity has been exercised, and with an encouraging degree of success. Personally, I shall always feel immensely indebted to the late Professor Zaaijer, of Leyden, who gave surgeons such great encouragement along these lines. But when I peruse the records of the numerous cases in which the plan has been tried, I cannot but wonder if other and simpler means might not have prevailed in a good many. One of my friends told me that he had seen no less than 11 patients in one hospital in Moscow who were all suffering from simple stricture of the œsophagus the result of swallowing caustic fluids in suicidal attempts, and in each case the surgeon in charge was attempting the formation of a new œsophagus. Surely, one can scarcely suppose that such a wholesale resort to this measure must be necessary, and I cannot but feel that more and more effort should be exercised in attempts to restore nature's path rather than to remedy the disability by a surgical exploit of such magnitude and risk.

The following case illustrates the difficulties that may be met with in dealing with non-malignant stenosis.

Congenital Stenosis—Gastrostomy at 3 Years—Repeated Recurrence after Dilatation through Œsophagoscope—Failure of Retrograde Dilatation—Death after Plastic Operation.—The patient was a male war baby, born in February of 1918. At a

month old he started vomiting all feeds, and there was very exalted visible peristalsis over the epigastric region. The case was diagnosed by an experienced pædiatric physician as an example of congenital hypertrophic stenosis of the pylorus. The condition gradually improved under treatment and no operation was carried out, but the patient was never quite well. At 1 year the weight was only 16 lb. 4 oz., and at 2 years 32 lb. At about this age "the pyloric condition appears to have improved, and the trouble transferred to the cardia". Regurgitation became a very serious matter and the child steadily went downhill. At 4 years of age matters became desperate, and as a life-saving measure I made a gastrostomy, using local anæsthesia. The child soon improved and began to swallow, but he was not normal, lived mostly on liquids and semi-solids, and was much worse at times. The gastrostomy was allowed to close after about eighteen months, and subsequently it was a struggle to get down sufficient nourishment and he was always hungry.

When the child was 7 years of age it became clear that the condition would not pass off spontaneously, and that some form of treatment by dilatation would be necessary. At this time radiography after an opaque meal showed a distinct narrowing at the junction of the middle with the lower third of the œsophagus. The opaque material passed very slowly through the narrowed area, and there was a little dilatation of the œsophagus above. The question of self-dilatation was discussed and tried, but both patient and parents had the greatest aversion to this method, and it was found impossible to get the necessary co-operation.

At 8 years of age, my colleague, Mr. Frank Wilson, first saw the boy, and under general anæsthesia passed the œsophagoscope and demonstrated what appeared to be a fibrous stricture at the lower end of the gullet. This was dilated with success and the boy was able to swallow quite well for some months, after which the stenosis began to recur and swallowing became more and more difficult, the dilatation having to be repeated, and always under general anæsthesia. In all this was done fifteen times—six times between the ages of 8 and 13, and nine times during the last two years of the boy's life.

When he was just over 13 he was in very poor condition and was practically living on milk. Although going to school, he was small and weedy and only weighed 5 st. 6 lb. At this time, a further effort was made to get the boy either to learn to pass bougies or at least to allow bougies to be used without general anæsthesia, but despite his miserable condition he could never be persuaded to make a determined attempt. As a rule he was very well after dilatation, but he did not always get the same amount of relief, and as time went on it became necessary to do this more often. The dilatation was sometimes followed by slight hæmorrhage, and during the last year of his life he had a very serious hæmorrhage which nearly proved fatal.

When he was just 14 it was found impossible to dilate the stricture, for the bougie appeared to impinge against something hard and fixed, rather like a new growth. Mr. Frank Wilson, who made the examination, came to the conclusion that it was a mass of inflammatory induration about the stricture, suggesting peri-œsophagitis, the result of chronic local infection, and that further attempts at instrumentation with bougies might result in a flare-up, with dire results. The boy himself said that he swallowed without much difficulty, but, as a matter of fact, it took him an hour or thereabouts to get down half a slice of bread with something to drink after each bite, and he was obviously going downhill. It was decided to attempt retrograde dilatation and to leave a rubber tube in situ.

With this in view, I first opened the stomach and attempted the passage of a bougie, but I found that the latter became entangled and I could not get it through. The cardiac opening, as felt from the stomach, was quite wide and easily admitted the forefinger, but a fold of soft mucous membrane, like a comb, could be felt anteriorly, and the actual stricture was just above this point. Mr. Frank Wilson now passed the œsophagoscope, and after several attempts with various sizes of bougies he finally managed to pass a fine whip. This went in so well that it was withdrawn in the hope of dilating the stricture up to a good size with a succession of instruments, but no other bougie could be passed, and it was only after several attempts that the whip was got through again. This time it was partly withdrawn from the stomach and a fine rubber tube was attached to it and was then drawn up through the mouth and left in situ. The operation was completed by remaking the gastrostomy. It was thought that the tube would act like a catheter tied through a stricture of the urethra and that it might be possible for the boy to swallow by the side of the tube. At all events, it was hoped that a bougie might be passed with much greater ease after the rubber tube had been kept

in situ for a week or so and, if that could be done, then it might be possible to teach the boy to use a small bougie. It was further hoped that the pressure of the rubber tube on the stricture might prevent the hæmorrhage which had occurred on recent occasions after dilatation. Unfortunately the tube was pulled out by the patient at the end of forty-eight hours. For a few days he did well, then had some hæmorrhage from the gastrostomy, but recovered and was able to return home in four weeks with the capacity for swallowing somewhat improved. After this he did try to pass a small Hurst's bougie, but it was shown by radiography to be doubling up in the œsophagus. The boy was better for a few weeks, when symptoms recurred rather suddenly and the problem was as before. Attempts were again made to use bougies without anæsthesia, but even faced with the fear of starvation the boy could not be induced to co-operate. Dilatation had again to be carried out under general anæsthesia, and it was after one of these attempts that a well-nigh fatal hæmorrhage occurred.

After due deliberation it was decided that the time had come for some more radical plan to be tried, and this seemed to hold out more promise since the stricture was known to be situated at the lower extremity of the œsophagus. It was hoped that either a cardioplasty or an œsophago-gastrostomy could be made, and it was decided to make the attempt. At the time the boy was in such poor condition that dilatation had to be carried out as a preliminary in the hope that he would pick up as a result and be in better condition for the major interference.

The following is the account of the operation as carried out on May 24, 1933, when the patient was just over 15 years of age: The stricture was dilated about three weeks previously and since then he had been taking his food very well, except for one day when it stuck just a little. The general condition was fair. He was given general anæsthesia by Dr. W. Seymour, and as a first stage Mr. F. Wilson introduced the œsophagoscope and used the evacuator, removing a considerable amount of thick mucus. After this it was easy to see the upper surface of the stricture, which had a sharp crescentic edge. The abdomen was then opened by the technique used for exposing the lower end of the œsophagus. The opening in the diaphragm was easily exposed and the lower part of the œsophagus explored. The latter looked perfectly normal and did not feel at all thickened, though an enlarged lymphatic gland was lying on its surface. Mr. Wilson now passed a bougie by the mouth and I could feel the end projecting into the œsophagus just above the cardia, but at this point it met with an obstruction and could not be farther advanced. The stomach was then opened and the finger introduced into the œsophageal orifice, where it encountered a stricture about an inch above the cardia. The stenosis was found to be about 1 in. in linear extent, and to have divided it completely would have meant incising the œsophagus up to a point at which it would certainly have been out of reach for suture. For the same reason it was not feasible to make an anastomosis between the œsophagus above the stricture and the cardiac end of the stomach, though that could have been done if the operation had been conducted through the chest. A vertical incision was made in the abdominal œsophagus below the stenosis and extended through the stricture for about three-quarters of its whole extent, but this meant a long incision extending well up into the mediastinum, and I was afraid to go farther. The remaining part of the constriction was completely avulsed with a bougie passed from above, then with a big pair of forceps from below, and finally with the Hurst's bougie passed from below. When this stage had been reached, there was a vertical incision through the lower end of the œsophagus 2 in. long and extending for another inch into the stomach. This appeared as a huge vertical slit, and it could only be stitched up transversely with very considerable difficulty, and not as securely as could have been desired. Some loose cellular tissue was fixed over the suture line and the œsophagus just above the upper end of the incision was stitched to the margin of the opening in the diaphragm. The liver was then replaced and a small tube brought from the site of the anastomosis out of the upper end of the parietal incision. The gastrostomy was again re-made.

The operation was very difficult and I was apprehensive about the result, as the suturing was not very satisfactory and leakage appeared likely. The whole proceeding took about one and a half hours. The patient was very well at the end and was quite well the same evening.

For two or three days he continued reasonably well and then the condition began to deteriorate. There was some hæmorrhage from the gastrostomy and he became anæmic, then some infection occurred around the tube and spread to the rest of the incision. Soon

after this the stomach leaked and this rapidly became serious. General sepsis supervened and he rapidly went downhill, dying on the twelfth day. There was no post-mortem examination.

In commenting upon this case and the unfortunate result, it occurs to me that it may be worth while mentioning the other schemes that might perhaps have been adopted. The first plan would have been to recognize temporary defeat and to have been content to remake the gastrostomy and to leave the problem of the treatment of the stricture until the patient arrived at an age at which he would be willing to submit to such treatment as required his intensive co-operation. Even if a subsequent operation was the only means of cure, then it would be much more likely to be successful with the patient in a better nutritional state as the result of the gastrostomy. Secondly, with a gastrostomy and the patient in good condition and in a co-operative state, it would have been possible to try again with the thread method or any other method that held out a prospect of success. If these methods failed, then, thirdly, we might have recognized that it was unwise to do anything more in the hope of dealing with the stricture directly, and might have been content to make an extrathoracic œsophagus.

I have since thought that in a case where the stenosis originated above the œsophageal opening and was of considerable linear extent, it might be possible to slit it up boldly with scissors and merely stitch the outer wall of the œsophagus together, not attempting to make an œsophagoplasty. I believe if gastrostomy was carried out and if the patient was not allowed to swallow anything for, say, a fortnight, that this might be satisfactory, but of course there would be the risk of septic infection of the mediastinum.

In conclusion, may I reiterate that I have only brought forward this subject because I am convinced that it is of general interest and cannot be relegated to any one department of practice. Some readers may find the recital of the cases just a little tedious, but on the other hand, those who are in search of detailed records often have difficulty in finding together such an illustrative series of the various types as I have been able to present. It is for that reason that I have thought it worth while to publish them fairly completely.

I am well aware that very many justifiable criticisms may be made, and would like to suggest that at some future time the subject might provide a useful discussion before our Society.

One lesson that I particularly want to draw from a consideration of the cases presented, is that we must not be too ready to conclude, when the difficulties of management are great, that the only solution, or the readiest solution, is the formation of a new œsophagus. This is a piece of reconstructive surgery of great magnitude, and while we cannot but admire the efforts of those who have carried cases to a successful conclusion, it ought to be known that there are a great many difficulties to be encountered and that the efforts of numerous surgeons the world over have been attended, not only with a high mortality, but with very many disappointments. No properly trained and experienced surgeon need hesitate to undertake the series of operations which are necessary for the construction of a new gullet, if this is the only means that will free the patient from such a terrible affliction as inability to swallow, but, after all, the restoration of this function by the normal channel is a better objective and a finer achievement.

I am very much indebted to those of my colleagues who have asked me to undertake the care of patients and have collaborated with me in their management, and I would especially mention Mr. Frank Wilson, Mr. Francis McGuckin, Dr. Horsley Drummond, and Mr. Munro Black, of Newcastle-upon-Tyne, and Mr. R. E. Jowett and Dr. James Waters, of Sunderland. I am also indebted to Sir Frederick Menzies, the chief Medical Officer of Health of the L.C.C., for permission to quote the cases which I have had the opportunity of treating at Hammersmith Hospital.

REFERENCES

[1] OCHSNER, ALTON, and OWENS, N., *Ann. of Surg.*, 1934, Dec., 1055.
[2] MORISON, RUTHERFORD, and DRUMMOND, HAMILTON, "Congenital Stricture of the Lower End of the Œsophagus", *Lancet*, 1913, i, 1021.
[3] LAMBERT, ADRIAN V. S., *Surg. Gynecol. and Obst.*, 1914, Jan., 1.
[4] TURNER, G. GREY, Henry Jacob Bigelow Lecture, *New Eng. Jour. Med.*, 1931, Oct. 1, 657.
[5] ROLLESTON, J. D., *Brit. Jour. Child. Dis.*, 1912, Jan., 12; *Ibid.*, 1913, Sept., 405.
[6] PRINGLE, J. H., STEWART, L. T., and TEACHER, J. H., "Digestion of the Œsophagus as a Cause of Post-operative and Other Forms of Hæmatemesis", *Jour. Pathol. and Bacteriol.*, 1921, xxiv, 396.
[7] KELLY, A. BROWN, "Some Œsophageal Affections in Young Children", *Jour. Laryngol. and Otol.*, 1936, li, Feb., 78; "Ascending Fibrosis of the Œsophagus in an Infant", *Ibid.*, 590.

HYPERTELORISM

NOTES ON THREE CASES

By H. P. PICKERILL, Wellington, N.Z.

Sufficiently few cases of hypertelorism have been published to warrant the recording of others that are observed, especially if they differ in important respects from those already published.

The word 'hypertelorism' has come to be applied to that form of craniofacial dysostosis showing an abnormal width between the eyes, sometimes, though not always, associated with other cranial or bodily defects or abnormalities. Garson Slo-Bodkin, in describing a case in the *Archives of Pediatrics* (1936), states that only 24 cases had been previously recorded. The condition was first described by Greig, of Edinburgh, in 1924. Before then I had seen 2 cases, but did not recognize them as clinical entities, considering them to be isolated abnormalities for which little or nothing could be done, and unfortunately I have no detailed notes or photographs of these two early cases. Thus, with the 3 cases to be described here, I have in the past seventeen years seen 5 cases of hypertelorism. This suggests that the condition may not be so extremely uncommon, but rather that the number of cases recognized has been few.

Greig (1924) regards the aetiology of the condition as residing in an overgrowth and early ossification of the lesser wings of the sphenoid, thus leading to a separation of the orbits, the fœtal lateral deviations of the orbit remaining fixed. Reilly (1931), after an exhaustive clinical examination of 4 cases, states: "There are no distinguishing laboratory signs, except that roentgen examination reveals the previously described skull characteristics. There are intersutural bones, internasal, parietomastoid, and pterionic, the sutures are incomplete, and there are no digitations." Dyspituitarism has been suggested as an underlying factor, but very few of the recorded cases show other definite signs of endocrine disorder.

Two other factors which are said to complete the syndrome are heredity or familial tendencies and distinct submentality, but again only a few of the cases recorded show both these factors.

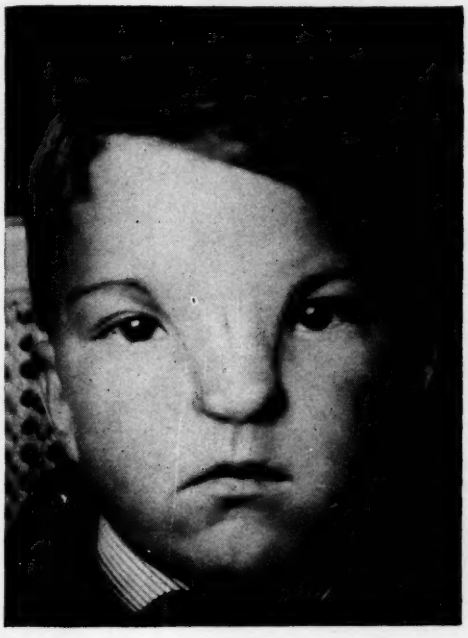
It has been said that simple hypertelorism is rare, but all the 5 cases I have seen have been such—at least there were no familial histories or submental symptoms. Other associated physical deformities may be syndactylism, large terminal phalanges, high palate, bifid uvula, undescended testicles, and congenital disorder of the heart. None of my cases has shown any of these factors of the syndrome, which makes one think that all these additional signs may be more likely associated with the submentality factor than the hypertelorism. In fact, that hypertelorism is not of necessity linked with mental defect, but may, like other physical defects, be sometimes associated with it.

Clifford Allen (1933–4), describing a case of hypertelorism in a woman suffering from amentia, epilepsy, and a paranoid psychosis, puts forward a theory that the condition of hypertelorism is due not primarily to a dysostosis but to aberrations

of brain growth in abnormal directions. One of my cases would appear to support this, in that there is an accompanying plagiocephaly, but this fails to explain the perfect symmetrical widening of the interorbital space. If the hypertelorism were due to the enlargement of one side of the brain one would expect the orbit on that side to be farther from the centre line than the one on the " normal " side, but this is not so ; in this case, as in all my cases, the orbits appear to be displaced outwards equally on each side.

CASE REPORTS

Case 1.—A boy aged 6 years. The patient was normal in every respect, except for the excessive width between the orbits, the width of the base of the nose, and distortion of the tip of the nose. It is some nine years since I first saw this patient, and he has now developed into a brilliant boy at school and is very good at sports. There is no family history of a similar deformity, and the mother, father, and sister are particularly bright mentally.



FIGS. 398, 399.—*Case* 1. Photographs showing the nasal deformity.

There are no other physical defects in the patient. The deformity was noticed at or soon after birth, which was an easy one and no instruments were used. The radiographs of the skull reveal no other abnormality than that the width between the orbits is excessive—the distance apart of the two internal canthi is just a shade under 2 in. The point of the nose is retracted irregularly upwards, the columella is undeveloped, the shape of the nose is that of a V in full face, and in profile is almost flat with the face (*Figs.* 398, 399).

Case 2.—A girl aged 13 years. This case is similar in almost all respects to *Case* 1. The deformity was noticed at birth, which was uncomplicated and no forceps were used. There is no family history of a similar deformity and no other physical defects. The child is mentally alert and up to her average at school. The radiograph of the skull reveals no abnormalities.

FIG. 400.—*Case* 2. Photograph showing distortion of the nose.

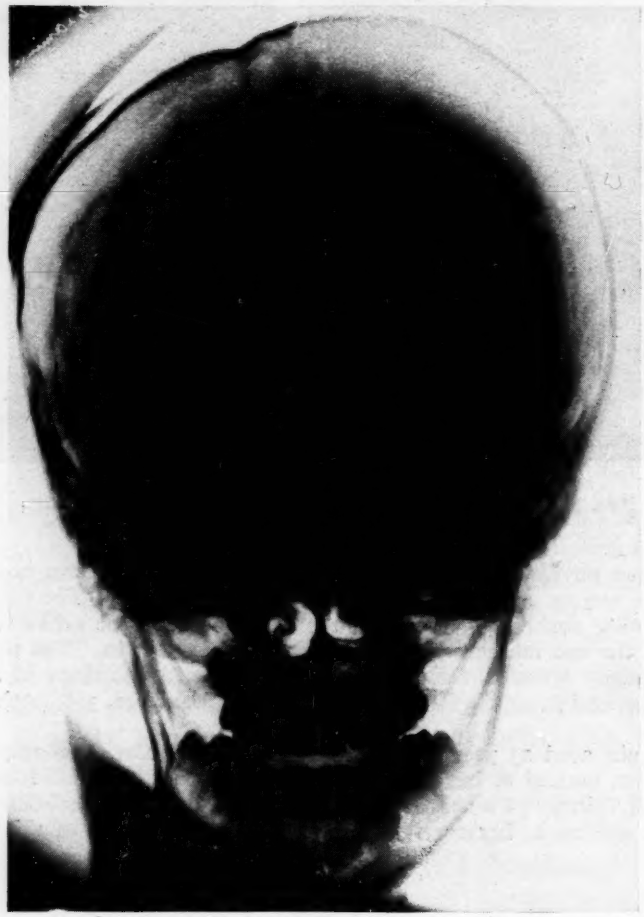


FIG. 401.—*Case* 3. Radiograph of the skull, showing a condition of plagiocephaly.

The width between the internal canthi across the bridge of the nose is 2 in. There is considerable retraction upwards of the point of the nose, and distortion upwards of the right nostril more than the left (*Fig.* 400). There is the same V-shaped nose in full face, but it is not quite so flat in profile as in *Case* 1 (*Fig.* 399).

Case 3.—A girl aged 16 years. Here also there is no family history, no history of damage at birth, no defective mentality, and no other physical deformities other than in the skull, which in this case, as may be seen in the accompanying radiograph (*Fig.* 401), shows a decided plagiocephaly. The skull as a whole is megalocephalic, the greatest circumference measuring 24 in. The width of the base of the nose between the canthi is 3 in. The point

FIG. 402.—*Case* 3. Photograph showing the patient after operation for restoration of the nasal septum and tip of the nose. The degree of hypertelorism can be well seen.

of the nose was drawn up, distorted, and dimpled in a manner similar to that seen in *Cases* 1 and 2, but unfortunately my photograph of this condition was a failure and the illustration seen in *Fig.* 402 is from a snapshot taken by her mother some months after I had operated to restore the columella and point of the nose, but it nevertheless illustrates the excessive hypertelorism quite well.

DISCUSSION

The two other cases which I saw some years ago were both in young women. They were both quite normal mentally and physically, and neither had any deformity of the point of the nose. They were referred to me simply on account of their hypertelorism.

It is difficult to realize that a premature ossification of the lesser wing of the sphenoid can give rise to these deformities, especially the excessive upward retraction and dimpling of the point of the nose. Clinically what impresses one is the massiveness of the nasal bones, and the lack of growth of the septum nasi.

Upper lateral nasal growth seems to occur at the expense of lower forward growth. The alæ nasi develop and grow, but the point of the nose gets left behind. In all radiographs of this condition a careful search has been made for any evidence of an interorbital cyst, but nothing of the sort has been found. The sella turcica has been normal in each case.

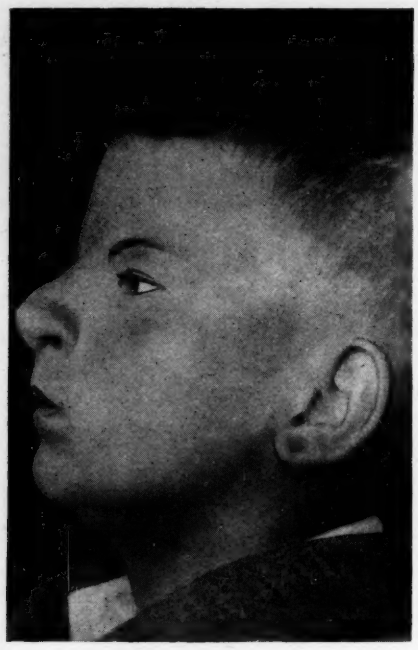


FIG. 403.—*Case* 1. Photograph showing the post-operative result. The bridge of the nose has been raised above the normal to allow for the subsequent growth of the rest of the face. (Cf. *Fig.* 399.)

Treatment.—Nothing so far as I know can be done surgically for the hypertelorism *per se*, but much may be done to improve the appearance of the patient by lengthening the columella and raising the bridge of the nose by cartilage grafting, as may be seen by reference to *Figs.* 402, 403. In *Fig.* 403 the bridge looks perhaps too much raised, but this has been done purposely to allow for the growth of the rest of the face, which in a few years will have developed to make the appearance normal.

THE HYPEROSTOSES IN RELATION WITH THE MENINGIOMAS *

By G. F. ROWBOTHAM, Manchester

The present communication is an inquiry into the nature of the changes in the cranial bones overlying the meningiomas, and especially with invasive and hyperostotic alterations. This is a fitting subject for inquiry because not only do these changes vary from case to case, but they may be absent; it is, therefore, of no small interest to inquire why this should be so; and in tracing the nature of the osseous changes, to inquire into their variations and to establish on a moderately large series of cases their clinical, no less than their pathological, appearance. This review is based on 100 cases of meningioma, of which 23 showed changes appropriate to this discussion. The neuro-surgeon and the neurologist have come to expect changes in the cranial bones overlying meningiomas—changes which should be recognizable radiologically. But these architectural alterations are more frequently produced by the enlargement of the vascular pattern of a larger or smaller area of the skull than by actual neoplastic invasion. This question of the circulatory alterations induced by these tumours will also be discussed.

HISTORICAL SURVEY

Though swellings on the skull are probably as ancient as man himself, it was not until the beginning of the nineteenth century that a serious attack was made on the details of their pathology. As a result, the fact has emerged that a large number of tumours apparently of the skull, whether they be small or large, begin from a primary pathological focus in the meninges. This opinion was not easily arrived at, for there were not wanting anthorities who held that the tumour in the bone induced a tumour in the brain either by spread or by nervous influences, and the history of the subject has shown that opinion has swung now this way and now that.

In the papers of Matthew Baillie, M.D., arranged by James Wardrop (1825), there is a short section dealing with the diseases of the brain and its membranes, Baillie stating, without expressing an opinion, that: "Bony tumours are sometimes found in the cranium, which press on a part of the brain. They most commonly consist of an irregular mass which is formed of bony processes with a fleshy substance filling up the interstices between them".

Pathologists of eminence, such as Rokitansky, Cruveilhier, and Wedl, in the early nineteenth century discussed the pathology of tumours of the dura mater under the old-established term, 'fungus duræ matris', and noted that such tumours occasionally invaded the skull. This is in accord with modern opinion, except that the authors did not understand the true nature of the tumour.

* From the Neuro-surgical Service of the Manchester Royal Infirmary. This study was aided by a grant from the Dickinson Scholarship Fund, and formed the basis of a Hunterian Lecture in 1935.

Although Lebert (1861) in his *Atlas of Pathological Anatomy* showed an excellent drawing of an enostosis found in relationship with a ' dural fungus ', and in an enlargement illustrates with great clarity the disposition of new bone built up in projecting columns from the inner table, he made little useful comment. John Cleland (1864), an anatomy demonstrator in Glasgow, described in detail two tumours found in the dissecting and post-mortem rooms growing from the dura mater, and noted the similarity of their histological structure to that of the Pacchionian bodies. One of these tumours lay close to the crista galli, and caused a thickening of the underlying wing of the sphenoid bone. This is not only the first record of a basal meningiomatous hyperostosis, but is the first suggestion of the origin of the cells which caused the tumour, ante-dating by nearly fifty years Mallory's recognition of the similarity of an arachnoid and a meningioma cell. In *Die krankhaften Geschwülste* (1864) Virchow described a tumour of the meninges which he called " Psammom der Dura Mater ", and noted that neoplasms of this type are occasionally associated with proliferative changes in the overlying bone and showed a diagram of an enostosis lying beneath an olfactory groove meningioma. Drummond, of Newcastle (1883), wrote a short paper on the diagnosis of proliferating tumours of the dura mater. Four cases were described, two of which were verified histologically and diagnosed as sarcomas, and he recorded examples by Chopart, Louis, Dupuytren, Cruveilhier, and Holmes. Drummond was particularly interested in the eye signs, and made a most astute remark on the presence of what was then called papillitis (papillœdema), which he discovered in all his four cases. He said : " Its importance I believe cannot be overestimated : and I trust observers will in future give attention to this point ". Brissaud and Lereboullet (1903) discussed a condition they called " hemicraniosis ", and described two cases in which there was a large swelling of the frontal bone and to a lesser extent of the face and lower jaw. The disease, they thought, was centred in the dura mater and caused the bone to proliferate as long as it was able. Having exhausted the possibilities of bony reaction, it began to act intracranially, producing the intradural tumour.

Parhon and Goldstein (1903) and Parhon and Nadjede (1905) reported further cases of so-called ' hemicraniosis ', but it remained for Barling and Leith, of Birmingham, in 1906 to give for the first time full emphasis to the similarity of the cells in the dural tumour and those filling the bony spaces in the cranial swelling.

Spiller, of Philadelphia (1907), reported two cases which are of considerable interest, because in the first, which he described before a local society in 1899, the bone was invaded by tumour cells, whilst in the second it was not. Evidently Spiller clearly understood that a hyperostosis could arise by invasion from within, but he was wrong in assuming he was the first to report such a case. The absence of tumour cells in the hyperostosis caused no particular comment. It is, however, of great interest, and this matter will be discussed later in the present contribution.

The radiological paper of Heuer and Dandy followed in 1916, but it was Harvey Cushing (1922) who made the first really authoritative statement on a sufficiently large number of cases. Up to that time most observers had had to content themselves with a report on one or two examples. Cushing's series of 80 cases had been associated with a cranial hyperostosis in 20 patients. He believed the bony thickening was caused by invasion of meningioma cells, with the resulting stimulation of osteoblasts and the production of new bone. About the same time, and independently, Wilder Penfield, working in the laboratories of Queen Square, reviewed 10

cases which had been operated on by Horsley and Sargent; this was the first communication which described in some detail the minute structure of the tumorous hyperostosis. Penfield pointed out that Gowers and Horsley had already appreciated the significance of swellings of the skull, knowing that some were merely the external sign of a deeply seated dural neoplasm. It was due to this perspicacity that Horsley operated on a nurse who had a swelling on the skull, and removed a dural tumour before the stage when increased intracranial pressure or neural dysfunction had developed. These two papers by Cushing and Penfield are outstanding communications.

Phemister in 1923 described 3 hyperostoses and, stressing the presence of meningioma cells within the bony spaces, believed that the new bone was not an integral part of the tumour but was reactive in nature, which was also the view of Harvey Cushing and Penfield.

Further pathological evidence rather along the same lines was adduced by Zachary Cope (1924) and Julian Taylor (1928). The latter pointed out that right-angled spiculation is not specific of the meningiomas, but also occurs in periosteal sarcomas and sometimes in secondary carcinomatous deposits of the hypertrophic type.

Kolodny (1929) put forward a new idea—he suggested that hyperostosis formation is due to the stimulus of the dilating vascular channels in the bone, and not to invading meningioma cells.

In 1934 Francis Echlin reviewed 18 cases from the literature, to which he added his own observations made on 9 specimens found in the laboratories of the Johns Hopkins Hospital. He compared the pictures given by the osteomas and meningiomas, finding that right-angled spiculation and intracranial encroachment were characteristics of the latter and rarely occurred in the former.

In recent years knowledge has grown apace and is widely diffused among the modern school of neuro-surgery, and recent communications on the radiological features of the meningiomas have come from Cairns, Twining, and Jefferson. Since this paper was written Harvey Cushing's monograph on the meningiomas has appeared. The reader will find there the early history of these tumours, often illustrated with copies of the original plates. Many of these primitive cases are grotesques.

MATERIAL

In a series of 100 meningiomas a hyperostosis was discovered on 23 occasions: 19 of these were associated with a meningioma of the massive type, and 4 with the flat or *en plaque* variety. There were 10 cases in men and 13 in women, and the patients' ages ranged from 26 to 70 years. The bones of the anterior half of the skull were affected in the very large majority of cases; in fact, the occipital bone was concerned on one occasion only—there was in this case a small enostosis in the groove made by the lateral sinus. The age of the meningiomas, as judged by the length of clinical history and by the number of years the hyperostosis had been known to have existed, is given in the appended table, along with clinical and histological details. All these cases have been under the care of Mr. G. Jefferson.

(Since the completion of this paper three further hyperostoses have been collected.)

ANALYSIS OF 23 CASES OF HYPEROST

No.	Clinical Details				Details of the Meningiomas			
	Sex	Age	Proteins in Cerebrospinal Fluid	Signs of Intracranial Encroachments	Position	Age as Judged by History	Anatomical Type	Cell Type
		yr.				yr.		
1	F.	34	35 mg.	Nil	Straddling ant. end of sup. long. sinus	1–5	Globoid	Psammoma
2	M.	28	Not known	Yes	L. frontal convexity	10	Massive	Meningotheli
3	M.	27	Not known	Yes	L. parietal parasagittal	1	Massive	Meningotheli
4	F.	60	Raised	Nil	L. frontal convexity	19	Flat	Meningotheli
5	M.	26	Not known	Yes	R. parietal parasagittal	3	Massive	Psammoma
6	F.	35	90 mg.	Yes	R. convexity	2	Massive	Psammoma
7	F.	43	450 mg.	Yes	R. sphenoidal wing	4	Massive	Fibroblastom
8	F.	46	60 mg.	Yes	L. basal and convexity	5	Flat	Psammoma
9	M.	42	Not known	Yes	Olfactory groove	2	Massive	Not known
10	F.	35	100 mg.	Yes	R. sphenoidal wing	3	Massive	Psammoma
11	F.	47	80 mg.	Nil	L. basal and convexity	20	Flat	Meningotheli
12	M.	70	Not known	Yes	R. parietal parasagittal	1½	Massive	Meningotheli
13	F.	54	75 mg.	Yes	Straddling ant. end of sup. long. sinus	3	Massive	Fibroblastom
14	M.	60	Not known	Yes	Occipital	8/12	Massive	Malignant sarcoma
15	M.	36	50 mg.	Yes	R. sphenoidal wing	9/12	Massive	Malignant sarcoma
16	M.	56	45 mg.	Yes	R. basal and convexity	1½	Flat	Psammoma
17	F.	30	20 mg.	Yes	R. parietal parasagittal	1	Massive	Fibroblastom
18	M.	60	120 mg.	Yes	L. parietal parasagittal	20	Massive	Psammoma
19	F.	42	190 mg.	Yes	Lesser wing r. sphenoid bone	3	Massive	Angioblastom
20	F.	32	Not known	Yes	Occipital	1	Massive	Not known
21	F.	38	220 mg.	Yes	Olfactory groove	—	Massive	Psammoma
22	F.	42	Not known	Yes	Convexity	5	Massive	Psammoma
23	M.	42	80 mg.	Yes	L. convexity	3	Massive	Fibroblastom

OCCURRING IN 100 CASES OF MENINGIOMAS

Details of the Hyperostoses				Operation	Result
Position	Age	Pathological Type	Clinical Type		
idline frontal	yr. 1–5	Diffuse tumorous	Swelling on skull	Removal	Cured
frontal and rietal	10	Diffuse tumorous	Swelling on skull	Three-stage removal	Died third stage
parietal	1	Diffuse tumorous	Swelling on skull	Removal	Cured
frontal and rietal	19	Diffuse tumorous	Swelling on skull	Biopsy	No change
idline parietal	3	Diffuse tumorous	Swelling on skull	Museum specimen	—
frontal and rietal	2	Diffuse tumorous	Swelling on skull	Removal	Cured
sal r. sphenoidal ing	4	Diffuse tumorous	Exophthalmos	Part removal	Improved
basal and tem-oral fossa	11	Diffuse tumorous	Swelling on skull and exophthalmos	Part removal	Improved
of of orbit	2	Diffuse tumorous	Exophthalmos	Part removal	Improved
sal r. sphenoidal ing	3	Diffuse tumorous	Exophthalmos	Removal	Cured
basal and tem-oral fossa	20	Diffuse tumorous	Exophthalmos	Biopsy and decom-pression	Improved
parietal	5	Osteoclastic	Swelling on skull	Removal	Cured
idline frontal	1½	Osteoclastic	Swelling on skull	Removal	Cured
cipital bone	8/12	Osteoclastic	Swelling on skull	Biopsy and decom-pression	Improved
mporal bone	1	Osteoclastic	Exophthalmos	Decompression and deep X rays	Improved
al r. sphenoidal ng	1½	Diffuse *non*-tu-morous	Exophthalmos	Removal	Cured
parietal	—	Nodal enostosis	—	Removal	Cured
parietal para-gittal	—	Nodal enostosis	—	Pathological specimen	—
enoid r. wing	—	Nodal enostosis	—	Removal	Cured
pital groove	—	Nodal enostosis	—	Decompression	Died
tory groove	—	Nodal enostosis	—	Removal	Died
rexity	—	Nodal enostosis	—	Removal	Cured
rexity	—	Nodal enostosis	—	Removal	Cured

THE PATHOLOGICAL FEATURES OF MENINGIOMAS

Meningioma is a non-committal term not implying a precise pathology. Much evidence supports the belief that the neoplasm arises in the primitive cell types of the arachnoid mater. In general these neoplasms are benign, rarely transgressing the limiting pial membrane and rarely exhibiting histological pictures of frank malignancy. The massive type, invaginating the pia mater, thrusts itself into the brain tissue and becomes to a greater or lesser degree buried. The meningioma *en plaque*, a much rarer type, is a sessile tumour of limited thickness growing along the planes of the meninges, in some cases for considerable distances. Both types may grow outward to invade the bone. These tumours vary considerably in vascularity and texture. Grossly, some are hard, others soft; the vascularity of the tumour itself varies considerably, and in some cases the hypervascularity of the surrounding structures makes the operative removal a most diffiult technical problem. In other cases the environments are relatively avascular. Further, the surface of the tumour may be smooth or extremely nodular, and the nodules themselves may be shaggy or smooth. That these variants have their histological counterpart was realized by Bailey and Bucy, who classified these tumours into nine groups. The psamomma, the most common type, consists of oval cells with densely staining nuclei, arranged in balls or whorls. Hyalin changes or calcium deposits commonly occur, forming the signatory psammoma bodies. The meningothelioma is a highly cellular tumour, with its cells arranged in sheets instead of in whorls. In the fibroblastic type the tumour is composed of fusiform cells arranged in bundles. It resembles a fibroma but for the frequent nests of endothelial cells. The angioblastic type is a highly vascular cellular tumour with a tendency to local malignancy, and its removal may present as formidable an operative problem as surgery can offer. The mesenchymal, lipomatous, osteoblastic, melanoblastic, and sarcomatous types are much less common, and their names are self-explanatory. Harvey Cushing and Eisenhardt have enlarged the grouping of the meningiomas, especially in separating twenty sub-species. However, Dorothy Russell and Bland, as the outcome of their studies on these tumours, have narrowed the classification to five groups, as tissue culture shows that the primitive cells are multipotential as regards eventual architecture.

THE CIRCULATION OF THE MENINGIOMAS

The vascularity of these tumours varies greatly, the hæmangioblastoma of the meninges (Greenfield) or the angiomatous meningioma (Bailey and Cushing), virtually a sponge of blood-vessels, being at one end of the scale, and the psammomatous variety, a relatively avascular tumour, at the other end. The arterial supply to the neoplasm is largely from the dura mater, though secondary communications develop from the internal carotid vessels in the pia, and cross to the tumour capsule. These are rarely large, but may be numerous; at operation most of the bleeding stops when the dura mater has been completely cut around the origin of the tumour. Towards the sagittal sinus the twigs of the meningeal vessels have become small, and it is rare to find a single large artery entering a meningioma. If this were so the operative problem would be easier. The bleeding met with during removal of these tumours is almost entirely venous or from indeterminate sinusoids. A striking feature is the richness of the circulation within the tumour and in the neighbouring structures. This commonly appears to be out of all proportion to the size and rate

of growth of the cell mass. The explanation probably lies in the primitive vessel pattern lacking sympathetic vasoconstrictor control, and to the presence of sinusoids and abnormal communications between arteries and veins, producing a vascular bed with a very low endovascular resistance. An excess of blood therefore easily finds its way through the neoplasm because of the absence of the arteriolar and

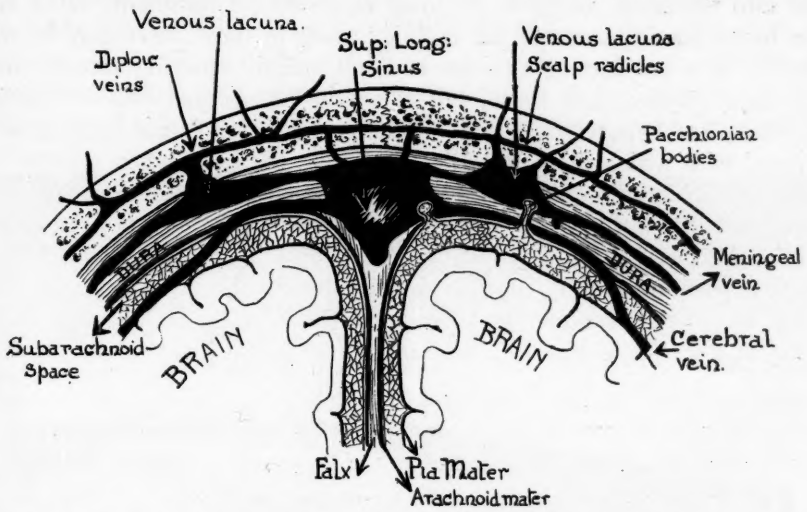


FIG. 404.—Drawing of the venous pathways draining the brain and dura mater, showing the channels which connect the diploic veins with the dural sinuses.

capillary resistance. This excess of blood has to get away, and it does so by enlarging and opening up new drainage channels. In the region of the superior longitudinal sinus the veins of the dura mater open into the lacunæ laterales and the veins of the cortex pass beneath the lacunæ to enter the main channel of the sinus itself (Le Gros Clark) (*Fig.* 404). There are important communications between the dural veins and those of the diploe, not only in the meningeal grooves but also in the many pits seen in the inner table near the middle line over the lacunæ. The diploic channels become dilated (*Fig.* 405), and it is this change which gives the radiographic picture so characteristic of the meningiomas. Jefferson thinks that the angioblastic meningiomas often write their signature in the bone. Large channels connect the diploic veins with those of the scalp, and by this circuitous route much of the blood circulating through the meningioma is drained.

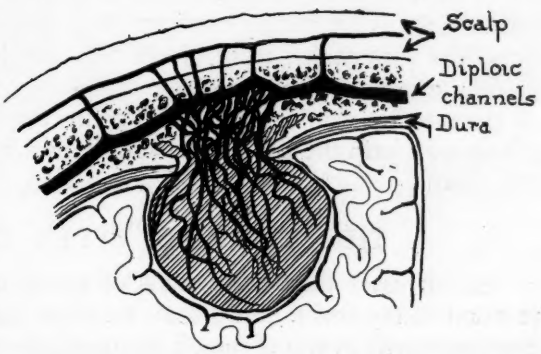


FIG. 405.—Drawing showing the increase, caused by a meningioma, of the venous channels which drain, from its dural attachment, into the diploic veins.

The importance of these unsuspected pathways in draining the brain has been demonstrated by Bedford, who ligatured the posterior part of the superior longitudinal sinus and found that the brain continued to drain adequately; on separating

the scalp from the bone the veins on the surface of the brain became congested, and serious bleeding developed from the scalp and skull surface. Such observations are only too often made at the operating table when the scalp is turned off the skull over bone altered by an underlying meningioma. Serious bleeding may at once arise, the bleeding points representing the greatly enlarged vessels which pass from the bone into the scalp, normally so small as to escape comment. It is true that the extra blood passing through the diploic vessels in these cases may be using the bone merely as a conducting channel and not strictly entering into its metabolic processes in a physiological sense, but it would be unreasonable to consider the bone anything but hypervascularized. Venous stagnation is not present, as shown by the speed with which thorotrast is cleared from the bone when injected into the external carotid circulation, a method which has been used on two occasions to gain further information on the circulation of meningiomas. In many of the cases in the whole series there was extensive dilatation of the diploic vessels without hyperostotic formation, which means that, though the circulatory change may be an important factor, it is not so important as the invasion of the bone by meningioma cells in the production of the hyperostoses. These cells were found in 21 of the 23 cases.

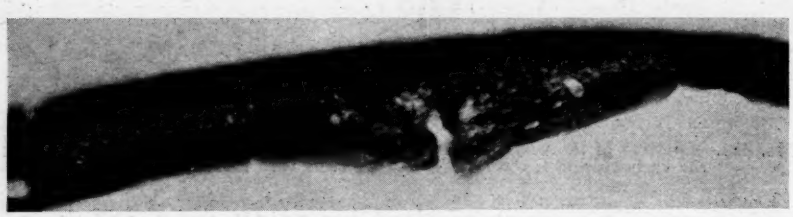


FIG. 406.—Radiograph of a nodal enostosis caused by the stripping of the periosteum by a large subendosteal venous lake through which the blood draining from the meningioma entered the diploic channels.

The chief part the circulation seems to play is to put the bone into a state of metabolic activity, and to break up its surface and enlarge the entering veins and so direct the current that meningioma cells are given the greatest mechanical advantage in their efforts to enter the bone. However, in spite of the important role played by the invasive meningioma cell, it seems certain than an enlarging sinusoid suitably placed may strip the periosteum and act as adequate stimulus to new-bone formation (*Fig.* 406).

THE CLINICAL TYPES OF HYPEROSTOSIS

In 20 cases there were signs of raised intracranial pressure or of local compression of the brain. In 3 cases only was there an absence of signs of intracranial encroachment; in two of these a flat meningioma was present, and in the third a small globoid tumour was found straddling the superior longitudinal sinus and indenting the relatively silent frontal lobes. The hyperostoses declared themselves clinically in three main ways: (1) Ten as an obvious swelling of the skull which could be easily seen or palpated; (2) Seven as an enostosis or mound of bone on the inner table demonstrable only by radiography; and (3) Six as a deformity of the orbit, with an accompanying exophthalmos or displacement of the eye.

1. Thickenings of the Vault.—Swellings on the skull are often not as simple as they appear superficially, and may have dangerous extensions beneath the surface.

They may be described as ' iceberg tumours '. Of the thickenings of the vault, the most typical form showed as a diffuse mass standing an inch or more above the surrounding bone. It was not confined to any single bone, because the suture lines did not act as limiting boundaries. Its surface, though undulant, was smooth, it had the consistency of the bone, and was never attached to the skin (*Fig.* 407). In one case the hyperostosis was small and occurred as a small hemispherical mound on the outer table of the parietal bone near the middle line (*Fig.* 408). It had all the external clinical features of a simple osteoma, though its true pathological nature was easily disclosed by the spiculated shadow shown in the radiographs, but more particularly by the Jacksonian attacks in the contralateral lower limb, which made

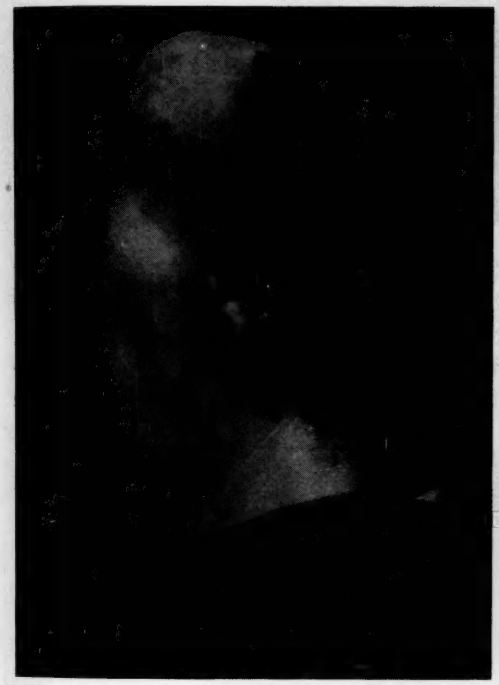


FIG. 407.—A large swelling on the left side of the skull caused by a diffuse tumorous hyperostosis.

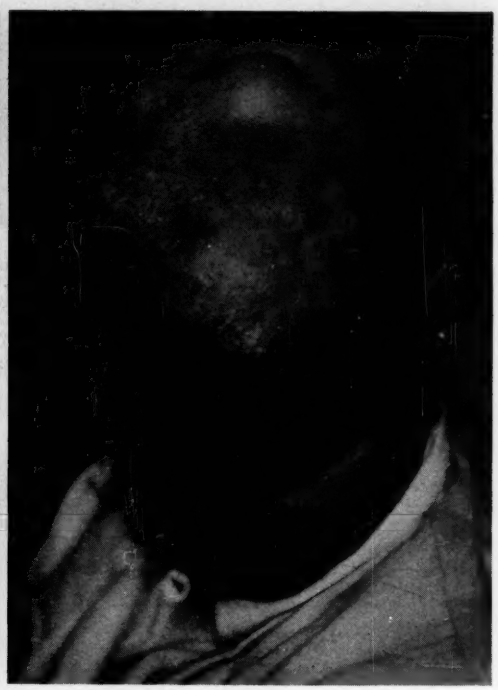


FIG. 408.—A small swelling on the left parietal bone overlying a parasagittal meningioma, causing convulsive seizures in the contralateral lower limb. Clinically the swelling closely simulated a simple osteoma.

so striking a declaration of its meaning. This picture, which can be used as the classic to illustrate the natural history of the meningiomas, both as regards neurological manifestation and pathological trend, is not in actual experience so common as some clinicians would have us believe; for, first, the tumour must be in exactly the right position, and, secondly, the bone must respond to form a local hyperostosis —two variables which coincide relatively infrequently. In our experience tumours have been opposite the foot area a few times, but in no other case was the bone involved in the same way as in the case illustrated.

2. The Enostoses (*Fig.* 409).—During the routine of radiography of the vault, parasagittal enostoses are frequently seen which are of no pathological significance. In the presence of positive clinical signs the enostosis is a most trustworthy aid in

determining the pathological nature of the neoplasm, for it is specific with meningioma. Special axial and stereoscopic views are often necessary to bring these small bony mounds into relief and to differentiate them from the normal markings of the frontal

FIG. 409.—Radiograph (antero-posterior view) showing a small nodal enostosis of the convexity.

fossa, or from the thickenings which so commonly occur in the region of the pterion. The diffuse nodal enostosis of the frontal bone (hyperostosis frontalis interna—*Fig.* 410) occurring chiefly in obese middle-aged women and not associated with an

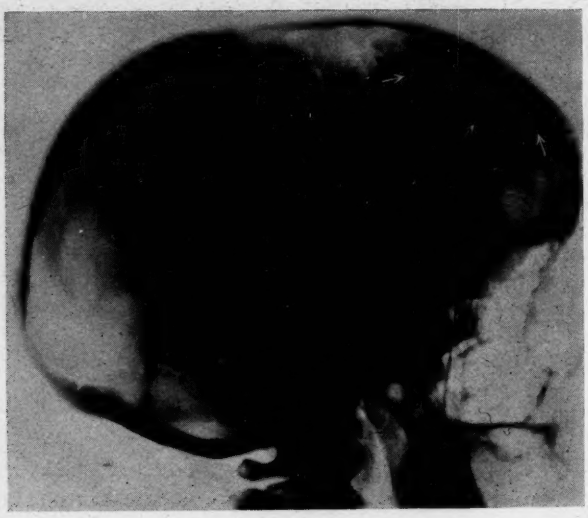


FIG. 410.—Radiograph of a 'hyperostosis frontalis interna'. This type of diffuse enostosis of the frontal bone is not associated with an underlying meningioma.

underlying neoplasm, is a well-known radiological entity and causes no difficulty in differential diagnosis. It is distributed diffusely over the inner surface of both frontal bones, it has a wavy outline with a sharp clearly demarcated edge, and is not accompanied by any abnormal vascular markings.

3. Displacement of the Eyeball (*Figs.* 411, 412).—Unilateral exophthalmos is by no means an uncommon clinical entity, and it may be produced by various neoplasms and inflammations. The diagnosis of an underlying meningioma depends on the presence of fullness in the temporal fossa, thickening of the sphenoid wings shown

FIG. 411.—Unilateral exophthalmos, with displacement of the eye, caused by a basal hyperostosis.

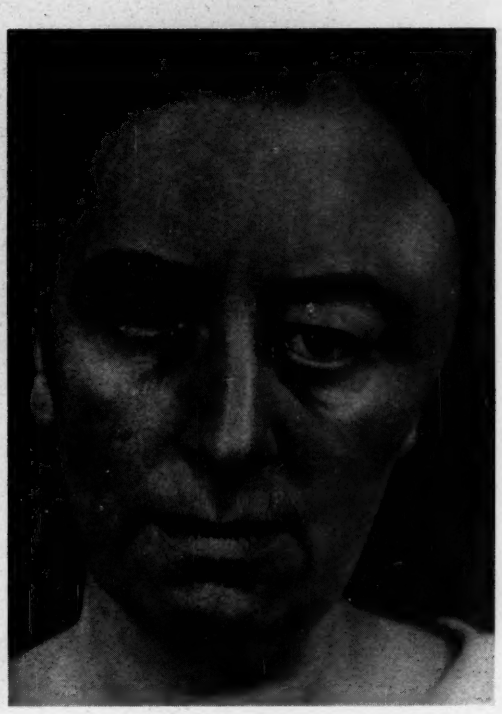


FIG. 412.—A swelling on the skull, with displacement of the eye, caused by a basal hyperostosis extending into the temporal fossa.

by radiographs, and an increase of the proteins in the cerebrospinal fluid. A considerable displacement of the axis of the eye may occur without causing diplopia, whereas the slightest interference with the neuromuscular mechanism leads to double vision. It is interesting to note that of the 6 cases of exophthalmos only 2 were caused by meningiomas *en plaque*, contrary to the well-established belief that the flat variety is a more common cause of displacement of the eye than is the massive variety.

PATHOLOGICAL FEATURES OF HYPEROSTOSIS

Hyperostosis, whether diffuse or nodal, seems to be peculiar to the meningiomas, for no other type of intracranial neoplasm has given rise to it. The alteration in the bone, whether diffuse or nodal, must of course be caused by the tumour, but the manner in which the hyperostosis comes about and the reasons why it is sometimes of one type and sometimes of another has never been explained or seriously discussed. In the approach to this problem it will be best to describe the bone changes as seen in typical examples and then to attempt some kind of synthesis. The main groups into which these cases have, for obvious reasons, been divided are set out below.

The Diffuse Tumorous Hyperostosis of the Vault.—In general, these hyperostoses were irregular fusiform thickenings, consisting of an enostosis and an exostosis, together with the original but altered tables of the skull (*Fig.* 413). The architectural pattern is best seen in the enlargement of radiographic pictures. Close to the tables bone is laid down in parallel trabeculæ and from these columns radiated inwards and outwards, the exostosis being capped with a thin layer of fluffy bone. The whole bony mass is extremely porous, with stout trabeculæ enclosing wide, relatively smooth-walled channels, in the lumina of which are masses of meningioma cells similar to those found in the intradural portion of the neoplasm.

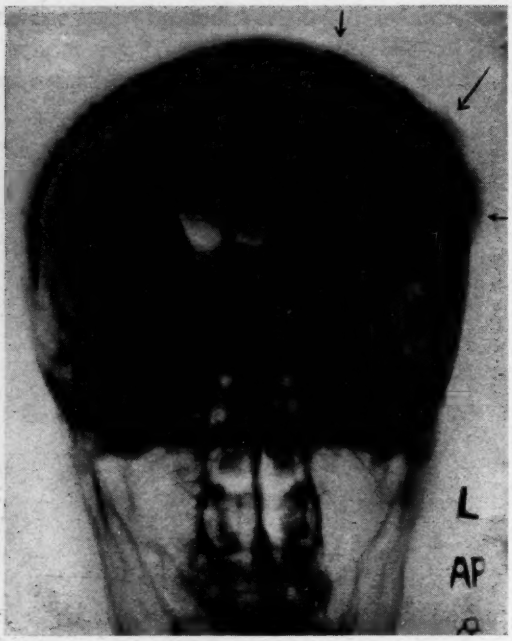
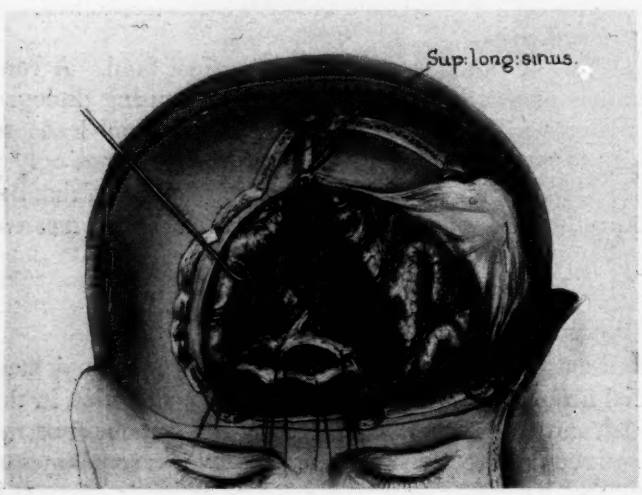
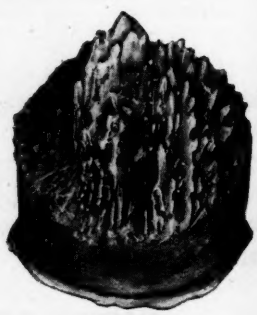


FIG. 413.—A radiograph (antero-posterior view) showing a diffuse tumorous hyperostosis. A ventriculogram had been done in this case. The ventricles are displaced to the opposite side.

There were 6 examples of diffuse thickening of the vault in which meningioma cells were found widespread between the trabeculæ of the hypertrophied bone. Of these, 5 were associated with meningiomas of the massive type and 1 with a meningioma *en plaque*, the tumour life-histories ranging from one to nineteen years. The hyperostoses themselves were known to have existed for periods of from two to nineteen years, and that their rate of growth was slow was shown in two cases which were observed for one and five years, during which time little appreciable growth took place.



A



B

FIG. 414.— A, Drawing showing the later stages of the removal of a meningioma straddling the superior longitudinal sinus. B, Showing the inner surface of a trephine disc taken from over the dural attachment of the tumour. Its inner surface is spiculated. The spicules were engaged by columns of meningioma cells.

The inner surface of the bone was invariably spiculated or eroded, and columns of cells continuous with the main meningioma mass plugged the pits in the bone, dovetailing the meningioma and the bone firmly together (*Fig.* 414). It is this pathological welding of the tumour to the skull which adds so many dangers and technical difficulties to the lifting of a bone-flap in these cases, as massive hæmorrhages can occur before an exposure has been obtained adequate to permit of control of the ruptured vessels.

In the literature of bone invasion by meningiomas the few examples described in any pathological detail have been drawn from this type of hyperostosis. There are equally important variants according to the degree of calvarial or of tumour dominance, and the two following cases have been selected to illustrate these types.

The first case, an example of calvarial dominance, occurred in a museum specimen in which the brain with its dural envelope, the meningioma, and overlying parietal hyperostosis were in situ. A study of this specimen was made possible by the kindness of Mr. A. R. D. Pattison.

The subject was a man, aged 26, who had had a swelling on the skull in the middle line, over both parietal bones, for two years, and who for one year had presented a progressive

FIG. 415.—Radiograph of a diffuse tumorous hyperostosis of the parietal bone. Both tables of the skull can be seen, and the diploic cavity is almost completely obliterated. The new bone, though showing evidence of right-angled spiculation, is laid down in trabeculæ parallel with the original tables of the skull.

right-sided hemiparesis with signs of raised intracranial pressure. The specimen was studied by the making of thin slices in the coronal plane with a circular saw. Examination of the transverse surface with a hand magnifying lens, immediately after section, showed the bone to be dense and the diploic cavity to have disappeared, apart from a few centrally placed streaks of pink. Radiological examinations were made of all the sections, and these gave the best pictures of the general architecture (*Fig.* 415). The original tables of the skull were quite distinct and had been so considerably thickened that the diploic cavity was almost completely obliterated. The new bone, though giving some suggestion of right-angled spiculation, had been laid down in layers parallel with the two tables of the skull, as though under the influence of factors governing the normal architecture of the calvarium.

The composite histological sections (*Fig.* 416) showed that the meningioma contained numerous whorls and was typical of the psammoma type. It was attached to the right wall of the superior longitudinal sinus and to the dura mater over the convexity for a distance of 1½ in. Meningioma cells were found in the lumen of the superior longitudinal sinus and within the meshes of its dural wall. From the upper surface of the sinus a vascular channel could be seen entering the bone, and along the course of this, either within its lumen or in its walls, meningioma cells could be clearly seen. The dura mater at the attachment of the neoplasm was split into fibres enclosing slit-like spaces, many of which were occupied by meningioma cells or filled with blood. The bone was extraordinarily porous in spite of its naked-eye appearance of compactness. In fact, multitudinous canals running throughout the

entire specimen formed the most noticeable histological feature. The internal surface of the bone was broken up with wide irregular pits and channels biting deeply into the bone. Radially directed channels spaced at intervals of one-eighth of an inch pierced the outer part of the hyperostosis for a third of its thickness. These openings gave the impression that they had been formed by an active process and not as a fortuitous arrangement, because the lamellæ in the bony columns had been laid down horizontally rather than vertically. Centrally the spaces tended to run in the long axis of the bone. Meningioma cells were found between the dura mater and the bone in the many pits on the inner surface of the hyperostosis and

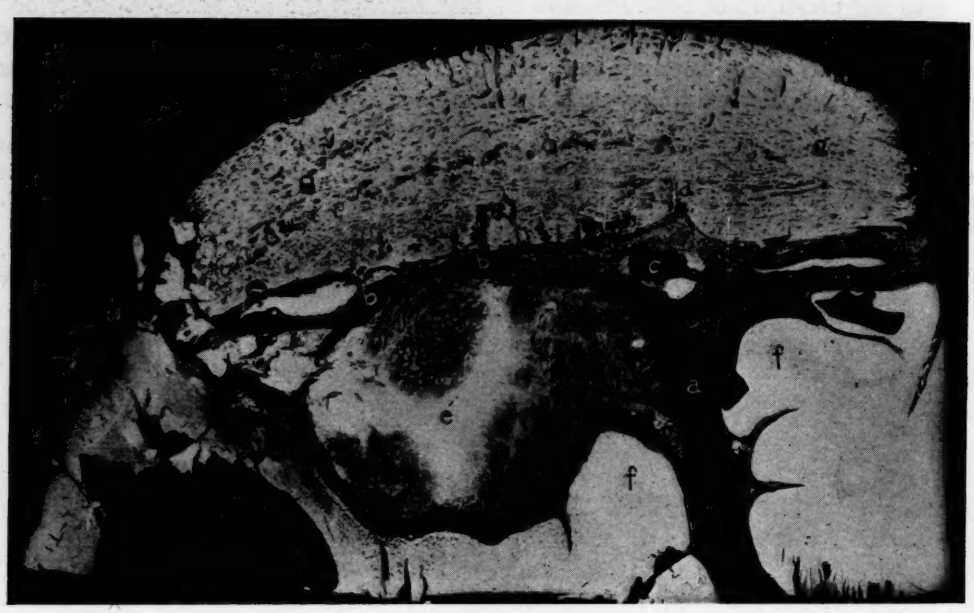


FIG. 416.—A composite histological section of a meningioma and hyperostosis in situ. *a*, Falx cerebri; *b*, Dura mater; *c*, Superior longitudinal sinus; *d*, Vein entering the bone; *e*, Meningioma; *f*, Cortex; *g*, Hyperostosis. Meningioma cells are present within the lumen of the superior longitudinal sinus, in the walls of the entering vein, and within the bony spaces.

in many of the more internally placed channels, though they were not diffusely distributed throughout the whole section as they were in the case next to be described. They appeared in clumps towards the centre of the lumen of the canals, and in places lined the margins of the bony trabeculæ. Many of the spaces were occupied by hæmatopoietic tissue and fibroblastic cells. Vascular channels whose walls were composed of a single layer of flattened cells were present amongst the neoplastic and fibroblastic tissues. Extremely few osteoclasts were found; indeed, rather less than the number of Howship's lacunæ would have suggested. Mononuclear cells similar to osteoblasts lined many of the bony spaces. On the outside the periosteum was thickened, and from its under surface prolongations dipped into the radially directed channels.

The second case, illustrating tumour dominance, occurred in a woman aged 34, who for one year had noticed a swelling in the middle line of the forehead which was slowly increasing in size and which was unassociated with symptoms and neurological signs of any kind. At operation a small globoid meningioma was found beneath this swelling and was removed. It had surrounded and completely obliterated the lumen of the superior longitudinal sinus. Masses of meningioma cells were found in the dura, and columns of cells could be seen invading the bone and filling the pits between the stout irregular spicules which projected from the inner table.

Histologically the bone was riddled with multitudinous spaces. From the outer surface large channels pierced the bone in a radial direction for a quarter of its thickness; towards

the centre the spaces were more irregularly shaped, the larger ones of which ran in the long axis of the bone, and the inner surface was broken up with deep pits and narrower channels tending to run towards the more centrally placed horizontal spaces. Meningioma cells were found in every part of the section and in fact were present in nearly all the bony channels and spaces. Hematopoietic elements were scarce, fibroblastic cells were numerous, and there were vascular channels with primitive walls composed of a single layer of flattened cells. The surfaces of the bony trabeculæ were lined by long spindle-shaped cells or by flattened cuboidal or columnar mononuclear cells resembling osteoblasts. In places giant polynuclear cells were present, as were Howship's lacunæ, in which such cells had presumably existed at an earlier period.

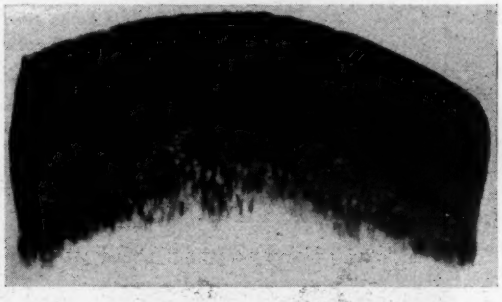


FIG. 417.—A radiograph of a diffuse tumorous hyperostosis of the frontal bone, showing tumour dominance. Both tables of the skull have disappeared, the whole architecture of the bone has been remodelled, and the bony trabeculæ can be seen projecting inwards and outwards.

Radiological studies (*Fig.* 417) showed that the whole architecture of the affected bone had been entirely remodelled. All traces of the diploic cavity and original tables of the skull had disappeared. The inner table had been replaced by spiculations, and radiating columns alternating with relatively clear spaces represented the remains of the outer table. This picture suggested that the new-bone formation was being dominated by the tumour invasion, and that calvarial influences were at a minimum.

Comment.—In the remaining four cases of diffuse thickening of the vault, the bone changes were similar in the main to those of the first case rather than the second. In these two examples the amount of bony new formation was remarkable considering the relatively short history of the cases, and remembering that some patients with longer histories show no sign of invasion at all. The type of the actual bone alteration was quite different in the two cases quoted. This might be due to the tumour on the one hand being histologically not the same, or to differences in the reactions of bone in different situations (for they were different) or in different individuals. The points will be further discussed later.

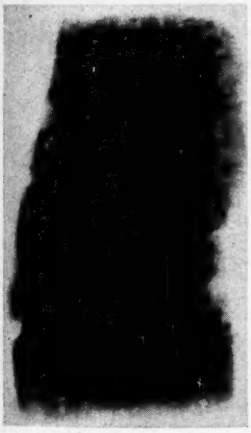


FIG. 418.—Radiograph of a diffuse tumorous hyperostosis of the great wing of the sphenoid bone, showing the irregular arrangement of the bony trabeculæ.

Diffuse Hyperostosis of the Basis Cranii.—It is quite certain that diffuse alterations can occur in the base of the skull no less than in the vault. They are not uncommon, and are chiefly associated with the olfactory-groove or sphenoidal-wing meningiomas. They may be the result of a globular meningioma, the common form of the parasagittal meningiomas, or the so-called meningioma *en plaque*, which is generally, though not invariably, a basal tumour. Its predilection for the region of the pterion is well known. In the present series the necessary piecemeal removal and the survival of all the cases has not given us the opportunity to examine a whole composite specimen as was the case in thickenings of the vault. However, in one case a large disc of bone was obtained from a central part of the hyperostosis which it is believed shows the typical structure of these hyperostoses (*Fig.* 418). It was obtained from a woman aged 47 in whom the hyperostosis was

known to have existed for 20 years. Comparative radiological studies over a period of seven years showed that the hyperostosis had grown extremely slowly, and in fact almost imperceptibly. In the past years the thickening of the bony walls of the

FIG. 419.—A radiograph of the optic canals. On the right side there are no abnormal changes. On the left side a basal hyperostosis has thickened the orbital walls. The orbital cavity is deformed and the sphenoidal fissure is narrowed.

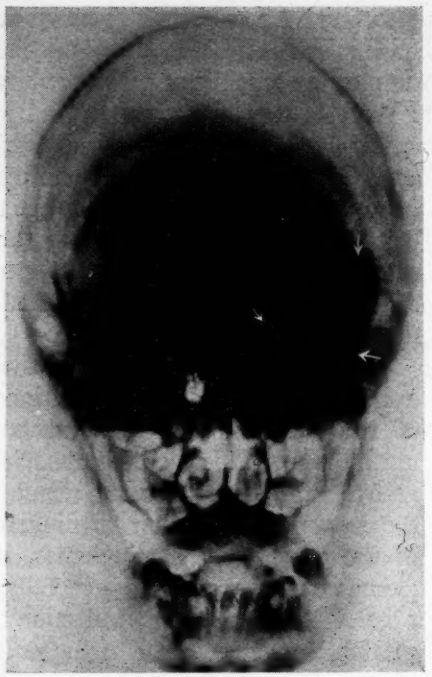


FIG. 420.—A radiograph (antero-posterior view) of a basal hyperostosis centred in the wings of the left sphenoid bone, causing deformity of the orbit.

orbit had so narrowed the cavity that it was found necessary to remove the eye (*Fig.* 419). At operation the scalp, temporal muscle, and outskirts of the hyperostosis were exceedingly vascular. The bone in the centre of the hyperostosis was

hard to cut and was relatively avascular. The underlying dura mater was lined with a diffuse welt of a meningioma *en plaque*. By the methods described in an earlier paragraph the structural details of the disc of bone were analysed. The bone was homogeneous, evidence of the tables of the skull had largely disappeared, and meningioma cells were found within the bony spaces. The basal thickening in all five cases was centred in the wings of the sphenoid bone (*Fig.* 420), and it is the impression that the sphenoid bone behaves in this way because of some special peculiarities of structure or of blood-supply.

Osteoclastic Hyperostoses (*Fig.* 421).—This term designates those rare cases in which the tumour has destroyed the overlying bone, the latter showing very little

FIG. 421.—A photograph of the inner surface of a bone flap showing bone erosion due to an underlying meningioma.

reaction and a minimum of resistance to meningiomatous invasion. Radiographically the bone has disappeared and there is a defect with a slightly irregular edge, suggesting a deposit of secondary carcinoma (*Fig.* 422). Tangential views (*Fig.* 423) show the bone to be expanded from within, and usually there is a very thin shell of external table covering them, though this may be incomplete. There were four such examples in the series, of which two call for comment. In one, a rubbery swelling under the scalp was situated over the left parietal bone close to the middle line. It consisted of the superficial part of a massive parasagittal meningioma which had invaded and destroyed the overlying parietal bone, pushing the periosteum outwards before it as the covering of a dome-shaped swelling. There were no abnormal vessels passing from the periosteum to the galea, nor were there any tumour cells in the subgaleal

space, the swelling being entirely subpericranial. The surrounding tissues were not particularly hypervascularized and the tumour itself was relatively avascular.

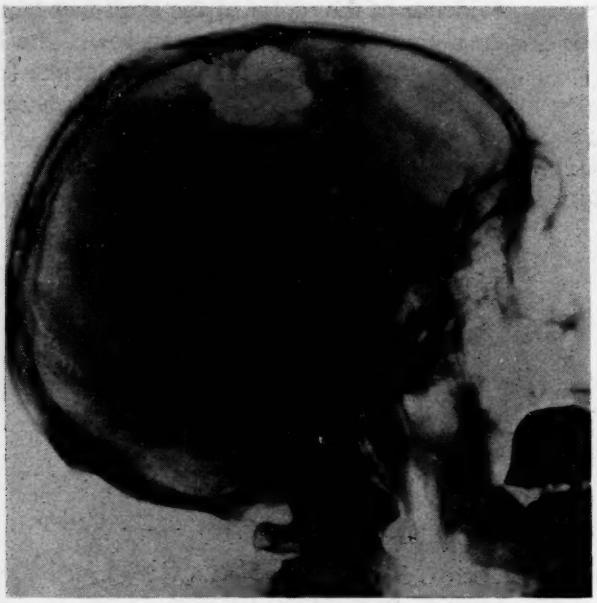


FIG. 422.—Radiograph (lateral view) showing the massive erosion of an osteoclastic hyperostosis.

FIG. 423.—Radiograph (lateral view) of a massive erosion caused by the invasion of a meningioma, showing a cyst-like dilatation of the frontal bone.

The bony defect was tightly packed with meningioma cells and its margins were shaggy and irregular. There was no evidence of the slightest effort at new-bone formation either in the invading tumour or in the skull itself.

In the second case there was a bony hard swelling on the forehead, centred a little to the right of the middle line (*Fig.* 424). Its diameter was 2½ in., it stood ¾ in. above the surrounding bone, and its lower margin was ½ in. above the supra-orbital ridge. A large parasagittal meningioma was found to have invaded both frontal bones, causing massive destruction with minimal new-bone formation. It indented the right frontal lobe, and its successful removal necessitated excision of a length of the superior longitudinal sinus. The two original tables of the skull had entirely disappeared, the bulging periosteum was lined with a thin shell of bone, and this cavity was plugged with meningioma cells. Pieces of bone nibbled from the margins of the defect showed extensive invasion of the bony spaces, with tumour

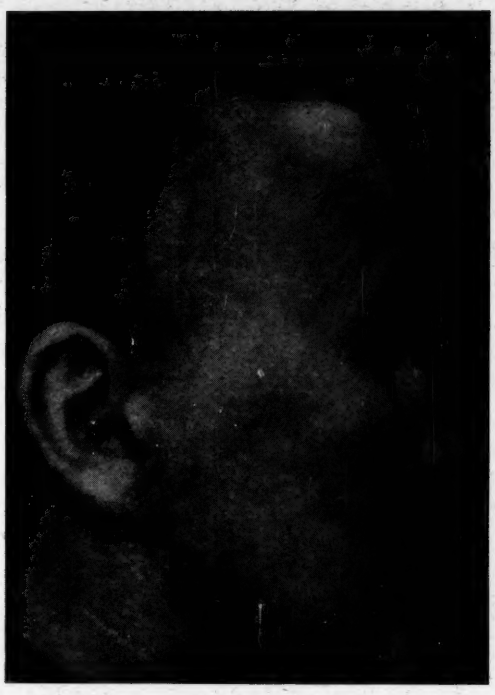


FIG. 424.—A large swelling on the frontal bone, caused by a meningioma which had eroded and ballooned the frontal bone.

cells and active osteoclasis in the walls of the trabeculæ. Both cases occurred in people advanced in years, the first in a man aged 70 and the second in a woman aged 54.

Nodal Enostoses.—Six nodal enostoses demonstrable by radiography were verified at operation and one at post-mortem. These circumscribed mounds of bone may develop from any of the bones of the vault or of the base. All these 7 cases overlay meningiomas of the massive type: 2 were found parasagittally, projecting inwards from the parietal bones over the dural attachments of meningiomas indenting respectively the right and left motor cortices; 2 overlay convexity meningiomas of the parietal lobe, their central point being 2 in. from the middle line; 1 was seen in the left olfactory groove; 1 straddled the impression of the left lateral sinus on the occipital bone; and 1 arose from the lesser wing of the sphenoid bone. When growing from the bones of the vault the nodal enostoses are hemispherical in shape,

measuring from ¾ in. to 1½ in. in diameter and projecting inwards for from ½ in. to ¾ in. They usually indent the centre of the dural attachment of the neoplasm. The enostoses growing from the base of the skull are usually not so symmetrical as those seen in the vault. They may cover a much larger area and consist of a group of conical projections standing up like stalagmites. Over one angioblastic meningioma the enostosis appeared as a framework of bone arranged round large vascular channels communicating with the diploic veins.

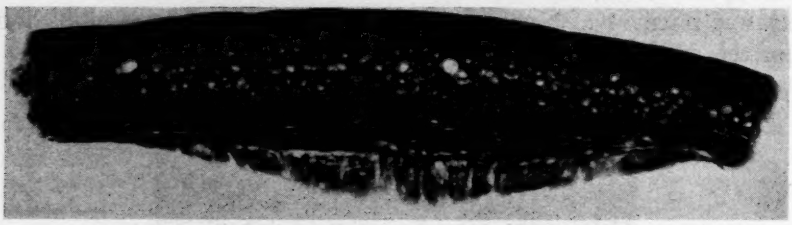


FIG. 425.—A radiograph of a nodal enostosis due to invasion of the bone by meningioma cells, showing the new bony trabeculæ arranged in right-angled spiculations projecting from the inner table of the skull.

Although at first sight the nodal enostoses give the impression of involving the bone locally but deeply, an examination of serial sections show that the enostosis is due to a deposit of new bone on the inner surface of the skull as if it were a periostitis. In one case, in a man aged 58, in whom a meningioma had existed presumably for twenty years, there was a small conical projection indenting the middle of the dural attachment of the meningioma. A wide-bored channel conveying a vein from the neoplasm to the bone ran through the centre of the enostosis and, from this, side channels were given off in many directions. The inner table was lifted and thickened as though the periosteum had been stripped and stimulated into activity. The new bone was laid down in layers parallel to the inner table (*Fig.* 406). Immediately

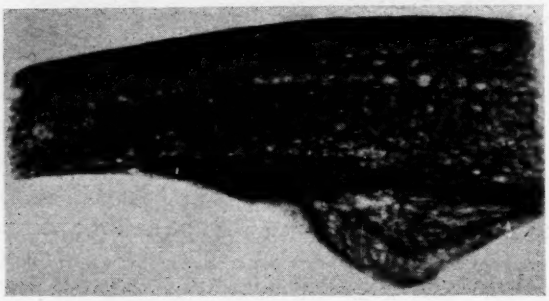


FIG. 426.—Radiograph of a nodal enostosis growing from the inner table of the skull, showing the new bony trabeculæ arranged as an irregular honeycomb.

surrounding the enostosis the face of the bone was broken up by the deep markings of enlarged dural vessels. In another case the area of local bone proliferation was made up of a palisade of bone fibres at right angles to the internal surface of the inner table of the skull (*Fig.* 425). These fibres were short at the edge and increased in length towards the centre, and were cemented together by transverse trabeculæ which were short and irregular and which did not traverse the whole diameter of

the enostosis. The inner surface presented a fine granular appearance which was due to the internally directed palisades, the bases of which were bound together by a compact layer continuous with the inner table. In this case the meningioma occurred in a young woman aged 30, and had given symptoms for one year only.

Between these two extremes of architectural pattern there are many varieties of internal structure. A third specimen illustrated this intervening stage very well indeed because the enostosis was made up of bony trabeculæ arranged like the irregular network of a sponge, the new trabeculæ of bone showing no tendency to be laid down either in radial or horizontal layers (*Fig.* 426). At the tip of the enostosis, and only at the tip, were found clumps of meningioma cells ; they occupied two

FIG. 427.—Microphotograph of a psammomatous meningioma. This neoplasm invaded the parietal bone and produced a massive diffuse hyperostosis. (Stain H. and E.) (× 475.)

bony spaces, they were not in intimate contact with the bone, and bone-cells, including osteoclasts, lined the walls of the spaces (*Fig.* 434). In none of the cases was there a diffuse ramification of the meningioma cells throughout the bony spaces as is usual in the diffuse tumorous hyperostosis.

It may be suggested that the enostosis is merely an early stage in the diffuse tumorous hyperostosis, but this is certainly not true of all cases, because ample time—five-and-twenty years in two cases had elapsed to allow of such development if it were going to take place. Cushing believes that new-bone formation associated with meningiomas is always due to invasion by tumour cells and these can be discovered if a careful enough search is made. An adequate search in three cases was made by serial sectioning. In two specimens a nest of meningioma cells was found within the bony spaces, and in the third specimen the whole block was examined

without the discovery of a single tumour cell. It is fully realized that in the drastic processes of decalcification of the enostosis small nests of tumour cells in the apex can be destroyed. Even so it seems likely that some of these local new-bone formations can be due to activation of the periosteum by stimuli independent of tumour-cell invasion. That enostosis formation is possible without tumour invasion is demonstrated in the cases of diffuse enostosis of the frontal bone, so well described in the literature (*see Fig.* 410). It is well known that in the presence of a meningioma the venous pathways between the dura mater and the bone usually become greatly enlarged and multiplied. If by chance a sinusoid be situated immediately beneath the endosteum, its enlargement will lead to local stripping of this membrane and so act as an adequate stimulus to new-bone formation, and when once this had been started, the drag of the meningioma by virtue of its weight and its movement caused by arterial pulsation will continue the process of separation. Such an arrangement of a subendosteal lake is shown in *Fig.* 406, and in this enostosis no tumour cells were found even after complete serial sectioning.

Diffuse Non-tumorous Hyperostoses.—There was one case in this series of a diffuse non-tumorous hyperostosis which gave rise to many interesting pathological speculations. Clinically, this case presented itself as a unilateral exophthalmos in a man aged 56 years, who had had symptoms of pain in the right eye and deterioration of vision in that eye for one and a half years. At operation a diffuse hyperostosis affecting the lesser and greater wings of the sphenoid bone was found, lying beneath a meningioma *en plaque*. It encroached on the orbital cavity, narrowed the sphenoidal fissure, and constricted the optic foramen to such an extent as to compress the optic nerve. The bone was an inch wide in the thickest portion of the hyperostosis, and was dense, hard to cut, and very avascular. The dura mater in the anterior and middle fossæ stripped from the hyperostosis with the ease met with in other cases in which the bone is not affected. No neoplastic tissue was seen outside the dura mater, and there were no abnormal vascular communications crossing the extradural space. The hyperostosis was completely removed and later not a single meningioma cell could be discovered within the bony spaces. It is possible that neoplastic invasion may have existed in some part of the hyperostosis and had escaped detection. However, what is certain is that large masses of bone can be produced, and that discoverable tumour cells no longer exist, suggesting that exciting mechanisms other than that of stimulation by tumour cells can play an important part in this type of hyperostosis.

THE RELATIVE IMPORTANCE OF THE PARTS PLAYED BY THE TYPE OF MENINGIOMA AND BY THE CALVARIUM IN THE FORMATION OF THE HYPEROSTOSIS

Reference has already been made to the gross morphological variations and differences of cell type which are to be found in the meningiomas. One must consider whether the reactions or lack of reaction in the bone overlying any given tumour might not be the reflection of the pathological difference between one tumour and another. That is to say, it might be the fact that a tumour with a particular cell type was especially prone to be associated with a special form of bony reaction.

The writer has inquired into this possibility and the present series seems to refute the idea. There are in the main four groups of hyperostosis and nine different kinds of meningiomas (Bailey and Bucy). Of the 23 meningiomas in the hyperostosis group, 21 were available for classification and 2 were not because there was an inadequate amount of material. Following the classification of Bailey and Bucy, 9 were of the psammoma type, 4 were fibroblastic, 5 were meningothelial, 1 was angioblastic, and 2 were frankly malignant or sarcomatous. In the 11 diffuse hyperostoses with neoplastic invasion, 5 were psammomatous, 4 meningotheliomatous, 1 was fibroblastic, and 1 unknown. In the 4 osteoclastic hyperostoses, 2 were sarcomatous, 1 fibroblastic, and 1 meningotheliomatous. The 7 nodal enostoses

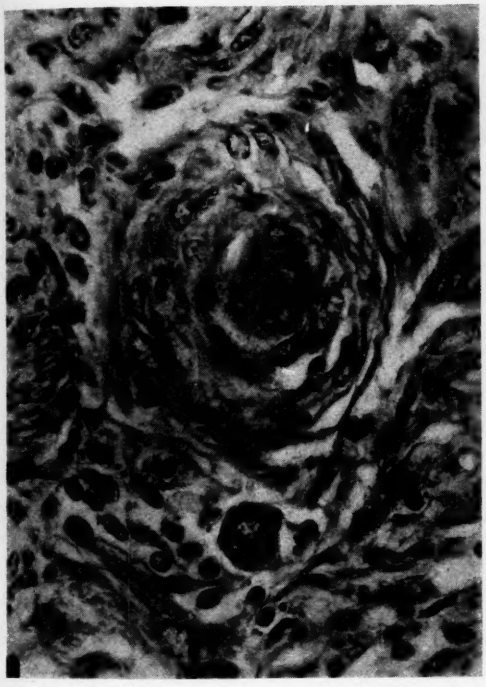


FIG. 428.—Microphotograph of a psammomatous meningioma. The cells with their large endothelial nuclei are arranged in whorls. Depositions of calcium can be seen dotted about the section. The neoplasm produced a small nodal enostosis on the convexity near the middle line. (Stain H. and E.) (× 475.)

FIG. 429.—Microphotograph of a psammomatous meningioma. This neoplasm did not produce a hyperostosis, and in fact failed to mark the internal surface of the bone by a single abnormal groove or pit. (Stain H. and E.) (× 475.)

were associated with 1 angioblastic, 2 fibroblastic, 3 psammomatous meningiomas, and 1 unknown. A psammoma overlay the basal diffuse non-tumorous hyperostosis.

From these figures it is clear that a large percentage of the meningiomas in the hyperostosis group are of the psammoma type, but this is also true of the whole series of meningiomas, with or without hyperostosis formation. It cannot therefore be confidently asserted that this type of neoplasm has particular osteogenic characteristics or particular tendencies to invade and alter the overlying skull. The most frank psammomatous types may cause a large diffuse tumorous hyperostosis (*Fig.* 427), a small localized enostosis (*Fig.* 428), or they may fail to make the slightest

impression on the bone (*Fig.* 429). Reference to the table (pp. 596, 597) will endorse this view, for we see that tumours with other cell types may produce precisely the same changes. Thus a highly cellular tumour of the meningothelial type, with its cells arranged in sheets, may cause a massive bone reaction (*Fig.* 430) or may destroy the overlying bone completely (*Fig.* 431). In two cases of massive bone destruction the cell types were entirely different, one was fibroblastic (*Fig.* 432) and the other meningothelial (*Fig.* 431). We must therefore conclude that no intimate relationship exists between the variety of the hyperostosis and the meningioma cell type. It seems, therefore, that the bone itself must play an important part in deciding the nature of the reaction, but before discussing the part the skull

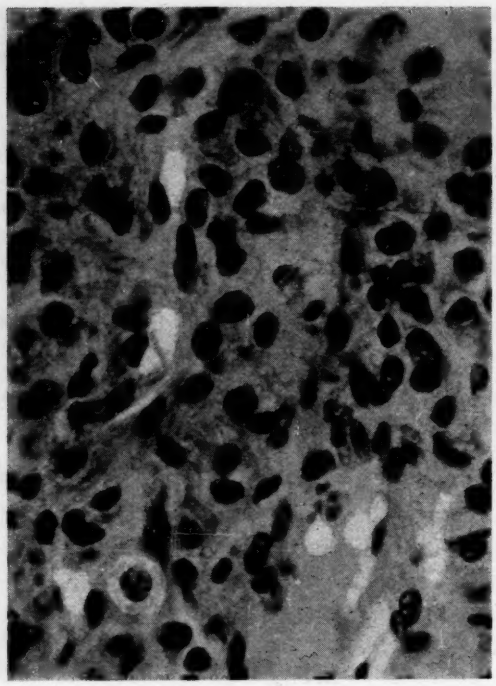


FIG. 430.—Microphotograph of a meningotheliomatous meningioma. The cells with their large endothelial nuclei are arranged in sheets and not in whorls as in the psammomatous types. This neoplasm invaded the frontal bone and produced a massive diffuse hyperostosis. (Stain H. and E.) (× 475.)

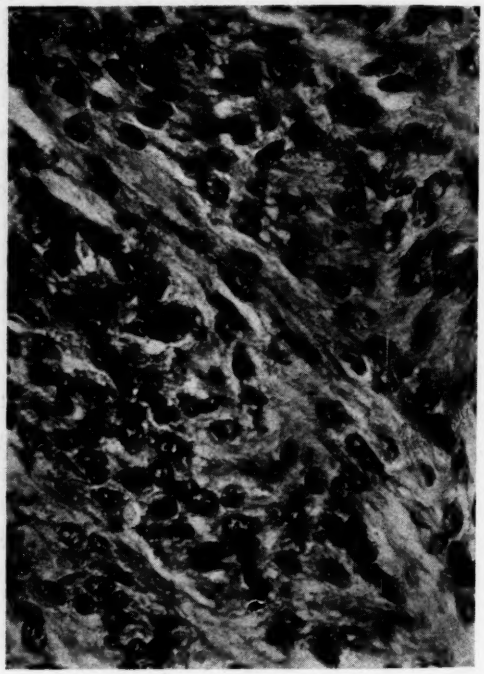


FIG. 431.—Microphotograph of a meningotheliomatous meningioma. This neoplasm invaded the parietal bone and caused massive destruction without stimulating any new or reactive bone formation. (Stain H. and E.) (× 475.)

plays in the hyperostosis formation, it is necessary to decide whether new-bone formation is an intrinsic part of the neoplasm, or not.

This important problem unfortunately cannot be settled by direct evidence, because microscopically the bone in the hyperostosis is indistinguishable from non-tumorous bone. This is a negative point, because bone can, though rarely, form in the middle of an intradural meningioma and yet have a normal adult structure. Comparative chemical analysis is fraught with too much error to be helpful in the solution of the problem, and metastatic bone has never been found as it sometimes is in cases of malignant bone-forming tumours in other parts of the skeleton, thereby indicating the osteogenetic properties of the cells in the primary tumour. However,

all the deductive evidence shows that the new bone in the hyperostosis develops from the bone of the skull and is not intrinsically neoplastic. If the new bone were neoplastic it would be difficult to explain the presence of what seems to be the original tables of the skull in many of the cases. In the two cases where the tables of the skull had disappeared, there was no bone found in the meningioma mass plugging the defect. Moreover, it seems strange that the extradural portion of these tumours should show such active new-bone formation when the intradural masses do not form a single bone cell (this point has been stressed by Penfield and Phemister). It has been suggested that the neoplasms may find origin in the dural, or in other words the periosteal, membrane and so the new-bone formation could be explained

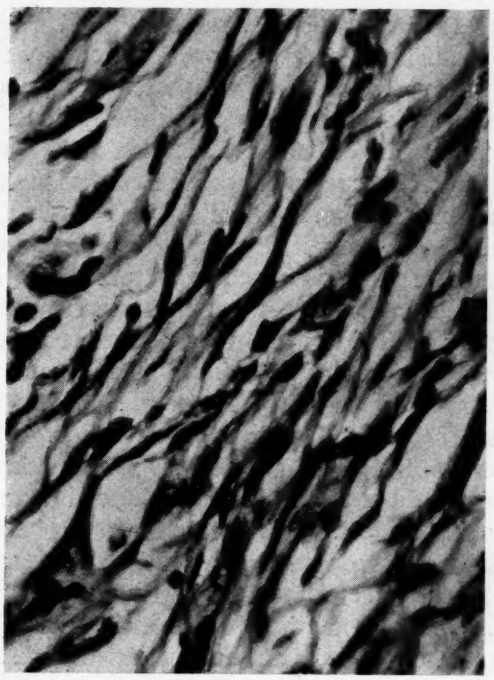


FIG. 432.—Microphotograph of a fibroblastic meningioma. The cells are spindle-shaped, with long flattened nuclei. They are arranged in bundles rather than in whorls. This neoplasm invaded the frontal bone and caused a massive erosion with the minimum reaction in new-bone formation. (Stain H. and E.) (× 475.)

in this way. Such an idea has recently been clearly stated by Globus in an interesting paper in which he has classified the meningiomas on the basis of the phylogeny and ontogeny of the meninges. Against this thesis is the overpowering argument that many of the hyperostotic tumours were not of the dural type, and that some of the most typical dural types have failed to reveal a single mark on the bone or to produce a single bone cell. Finally, there is much experimental work to show that non-osteogenetic neoplastic cells can excite a proliferative reaction in bone, and to support this evidence there are those cases of hypertrophic skeletal metastases which occur in non-osteogenic primary carcinomas, of which the ivory vertebra is an excellent example.

Therefore, if as we believe, the new-bone formation is due to reactive changes in the skull and is not an integral part of the tumour, the structure of the hyperostosis

will depend and vary, not only on the nature of the invading tumour, but on the properties of the bone affected. This was realized in the early stages, and so an investigation was made into the histology and architecture of the cranium in the various decades of life. Discs of bone were taken from corresponding sites from people of the same age and also of different ages and examined for histological detail. In brief, it was seen that the relative amounts of soft cancellous bone in the diploe and hard bone comprising the tables varied a good deal. In some the blood-forming elements were very active, in others rather quiescent; in some the diploic channels were abundant, and in others rather scarce. The thickness and vascularity of the periosteum varied, as did the ease with which the dura mater stripped from the bone. All these differences can be observed at the operating table.

It can be concluded, therefore, that a meningioma of whatever type will be in contact with bone of a different texture, form, and heredity from one case to the next. The nature of the hyperostosis depends, therefore, not merely on one variable, the tumour, but on two variables, the bone and the tumour. The final equation may be thrown over to the side of bony proliferation or of bone destruction. It is well known that osteogenesis is much more active in the young than the old, and it is significant that the most massive bone formation was seen in a young man, whereas the two cases of massive bone destruction associated with non-sarcomatous meningiomas occurred in people of more advanced years, one aged 54 the other aged 70. Thick bone of a dense texture seems particularly resistant to alteration or invasion, as many meningiomas have failed to make the slightest mark on the smooth polished internal surface of overlying bone of that kind. The age and size of the neoplasm itself have no special importance, save, of course, the obvious fact that the longer a neoplasm exists the more chances it has of invading the bone. Many massive tumours which have been known to have been growing for several years have failed to enter the bone. The extent of dural attachment may be of importance. For example, the plaque type of meningioma, with its diffuse infiltration along the planes of the dura mater, is more liable to invade the bone than the globular type with its localized dural attachment. No single factor can be shown to have dominance, all variables seem to be operating. In the bone, these are texture, vascularity, age, site; and in the neoplasm, cell type, extent of dural attachment, vascularity, age, size, and vascular connections with the bone.

ROUTE OF INVASION

How do the meningioma cells gain entrance into the bone and what is the nature of the stimulus which causes the bone to react? Meningioma cells were often found lining or plugging the venous sinuses. This is a well-known finding, and in Towne's case the tumour extended along the sinuses and veins as far as the superior vena cava. Within the neoplasms themselves could be seen spaces resembling blood channels or sinusoids whose walls were composed solely of a layer of flattened meningioma cells. In one specimen (*Fig.* 433) in which a composite section had been obtained of a hyperostosis with a parasagittal meningioma in situ, there were masses of cells in the lumen of the superior longitudinal sinus and also in the lumen of a vein leading at right angles from the upper surface of the sinus directly into the middle of the hyperostosis. That the veins are the main route of entrance of the meningioma cells into the bone there can be little doubt, and this is particularly so

in the early stages. As the face of the bone becomes broken up with vascular activity, it becomes easier for entrance by direct extension, especially as the dura will probably be in process of being invaded.

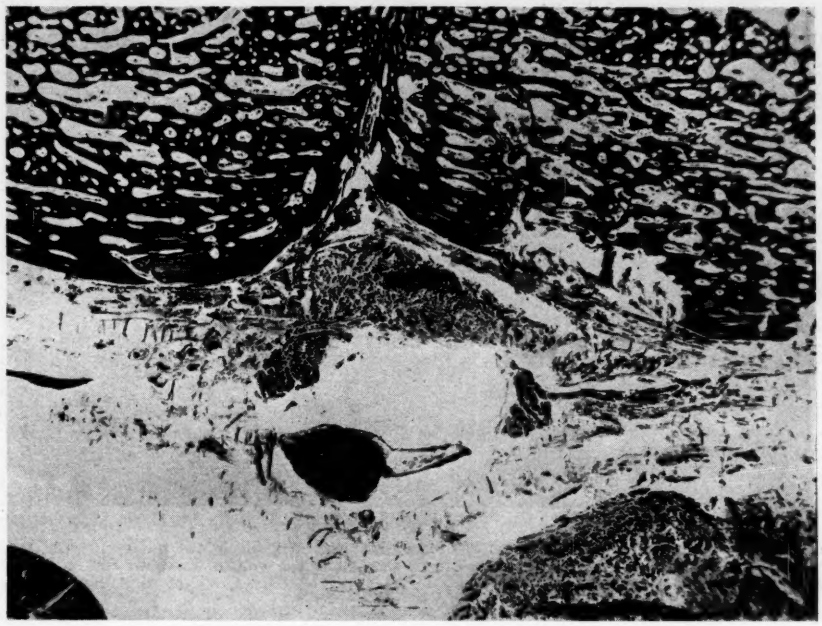


FIG. 433.—Microphotograph showing a vessel entering the bone from the top of the superior longitudinal sinus. Meningioma cells are present within the sinus and vessel. (× 6·6.)

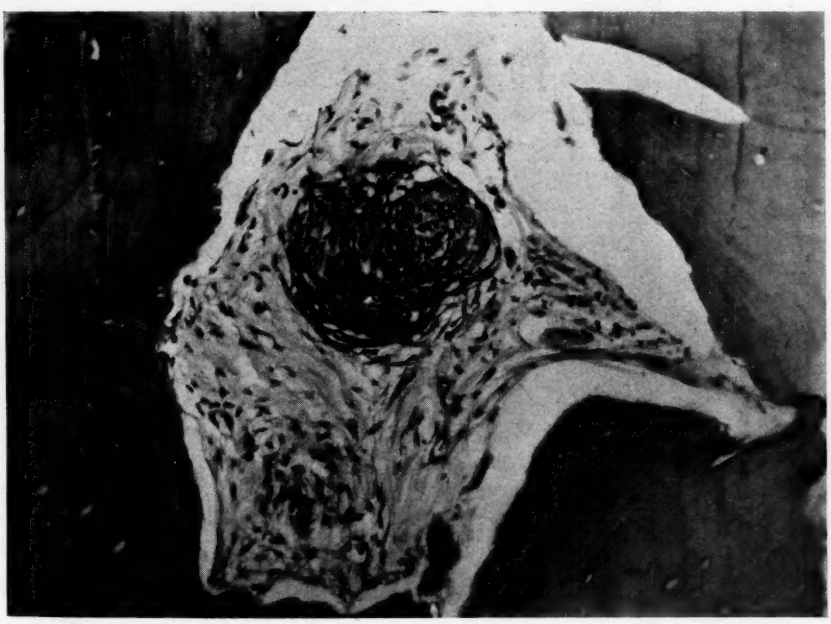


FIG. 434.—Microphotograph showing a clump of meningioma cells found in the tip of a nodal enostosis. Stain H. and E.) (× 166.)

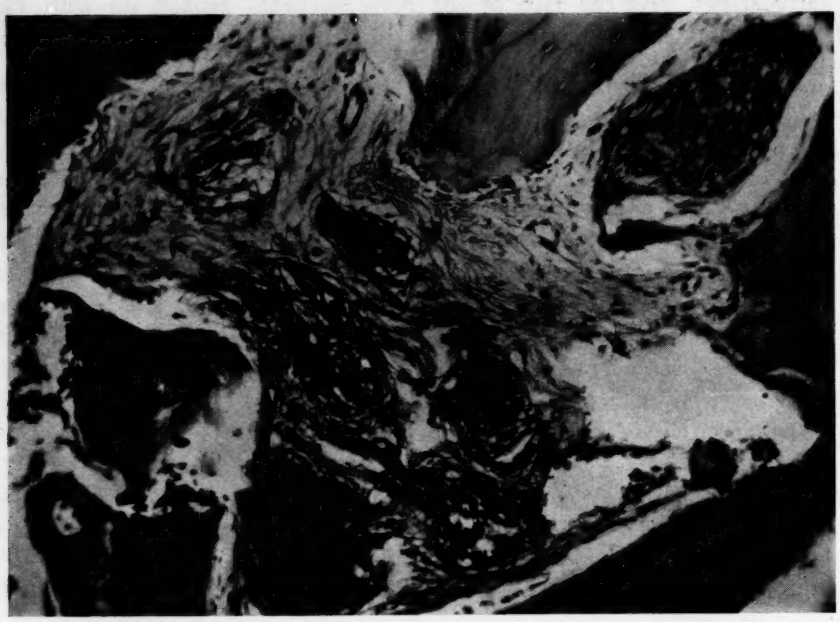


FIG. 435.—Microphotograph of groups of the meningioma cells within the bony spaces of a diffuse hyperostosis of the vault. Signs of osteoclasis can be seen along the walls of the bony trabeculæ. (Stain H. and E.) (× 133.)

FIG. 436.—Microphotograph of osteoclasts found at the edge of bone undergoing destruction in an osteoclastic hyperostosis. (Stain H. and E.) (× 475.)

In all but two cases meningioma cells were found within the bony spaces, in some they could be seen throughout the section, in others many of the spaces were occupied with blood-forming elements, and in one nodal enostosis only two small clumps of cells could be found within the very tip of the newly-formed bone (*Fig.* 434). The cells, accompanied by blood-vessels and fibrous elements, lay within large channels enclosed by the bony framework of the hyperostosis, they lay in clumps or lined the walls of these channels, but in no cases were they enmeshed in an intimate way within the actual structure of the bony trabeculæ (*Fig.* 435). In places large mononuclear cells resembling osteoblasts could be seen on the surfaces of the bony trabeculæ, with osteoclasts obviously eroding the adjacent bone. The general impression supports S. L. Baker's view that tumour cells do not act on bone directly, but rather set into action the intrinsic mechanisms of normal bone physiology. New formation is brought about by osteoblastic activity and erosion by osteoclasts (*Fig.* 436), activated in some unknown way by the tumour. Most of the new bone, as shown by the polarizing microscope, was of a mixed lamellar and 'woven' type, the lamellar type being rather more abundant than the woven.

Another question that should be put is this: are these invasions of the skull a sign of frank malignant intention on the part of the meningiomas? There is a good deal of evidence to support such a suggestion, as not only the bone but the muscle and the scalp may be invaded. Before accepting this suggestion, however, the general life-history of the hyperostoses must be considered, and in this group four had existed for four, ten, eleven, and nineteen years. Even when the hyperostosis was well developed the rate of growth was still slow, and one case has been observed for several years without any obvious increase in size being noted.

Judged on histological criteria, 19 out of the 21 cases were frankly benign and no metastatic deposits were found in a single case. Pathologically, the meningiomas in the hyperostosis group seem to possess certain undoubted malignant characteristics, but judged on life-history, freedom from invasion of the brain, and absence of metastatic deposits, they are essentially benign. There is no question, of course, but that these changes in the bone introduce technical difficulties into the operative procedures and make the removal of the meningioma more formidable.

This investigation was first suggested by Mr. Jefferson, and I would like to take this opportunity to express my gratitude for the very generous supply of his material, for his criticism of the scientific studies, and for his guidance in the production of this paper. Dr. Twining very kindly has shown an unceasing interest in the problems of this work, and to him I am also very grateful. I would also like to thank Miss Eugenie Cooper and Professor Baker for their careful criticisms of the histological conclusions. The excellence of the composite histological sections is due to the skill and care of Mr. H. C. Taylor, the chief technician of the pathological laboratories.

BIBLIOGRAPHY

ANDREWS, E. W., and ANDREWS, E., "Endothelioma of the Brain and Meninges", *Surg. Clin. N. Amer.*, 1923, iii, No. 4, 917.

ASHHURST, A. P. C., "Case of Jacksonian Epilepsy caused by Brain Tumour. Successful Removal of the Tumour", *Ann. of Surg.*, 1920, lxxii, 402.

BAILLIE, M., *The Works of Matthew Baillie, M.D.*, by JAMES WARDROP, 1825, ii, 387. London.
BALLANCE, Sir C., *Some Points in the Surgery of the Brain*, 1907. London.
BARLING, H. G., and LEITH, R. F. C., "Removal of a Cerebral Tumour (Endothelioma) which had involved the Overlying Cranial Bone", *Lancet*, 1906, ii, 282.
BEDFORD, Personal communication.
BRISSAUD and LEREBOULLET, P., "Deux Cas d'Hémicraniose", *Revue neurol.*, 1903, xi, 537.
CLARK, W. E. LE GROS, "On the Pacchionian Bodies", *Jour. of Anat.*, 1920–21, lv, 40.
CLELAND, J., "Description of Two Tumours adherent to the Deep Surface of the Dura-Mater", *Glasgow Med. Jour.*, 1864, xi, 148.
COPE, Z., "Case of Frontal Endothelioma with Hyperostosis Cranii (Pacchionian Tumour)", *Brit. Jour. Surg.*, 1925, xii, 794.
CRAIG, WINCHELL MCK., "Multiple Tumours of the Skull simulating Osteomyelitis", *Arch. of Neurol. and Psychiat.*, 1931, xxvi, 393.
CRAIG, WINCHELL MCK., "Malignant Intracranial Endotheliomata", *Surg., Gynecol. and Obst.*, 1927, xlv, 760.
CROWE, S. M., and JONES, C. K., "A Large Hyperostosis of Cranium due to Arachnoid Fibroblastoma", *Trans. Chicago Pathol. Soc.*, 1927, xii, June, 340.
CRUVEILHIER, J., *Traité d'Anatomie pathologique générale*, 1856, iii, 642.
CUSHING, HARVEY, "The Cranial Hyperostoses produced by Meningeal Endotheliomas", *Arch. of Neurol. and Psychiat.*, 1922, viii, 139.
CUSHING, HARVEY, "Experiences with Orbito-ethmoidal Osteomata having Intracranial Complications", *Surg. Gynecol. and Obst.*, 1927, June, 721.
CUSHING, HARVEY, "The Meningiomas (Dural Endotheliomas): Their Source and Favoured Seats of Origin", *Brain*, 1922, xlv, 282.
CUSHING, HARVEY, and WEED, L. H., "Studies on the Cerebrospinal Fluid and Its Pathway: IX, Calcareous and Osseous Deposits in the Arachnoidea", *Bull. Johns Hopkins Hosp.*, 1915, xxvi, 367.
DRUMMOND, D., "Note on the Diagnosis and Nature of So-called Perforatory Tumours of the Dura Mater", *Brit. Med. Jour.*, 1883, ii, 762.
ECHLIN, FRANCIS, "Cranial Osteomas and Hyperostoses Produced by Meningeal Fibroblastomas, A Clinical Pathologic Study", *Arch. of Surg.*, 1934, xxviii, Feb., 357.
ELSBERG, C. A., and SCHWARTZ, C. W., "Increased Cranial Vascularity in its Relation to Intracranial Disease", *Arch. of Neurol. and Psychiat.*, 1924, xi, 292.
EWING, JAMES, "A Review and Classification of Bone Sarcomas", *Arch. of Surg.*, 1922, iv, May, 485.
FÉRÉ, C., and ARNOULD, E., "Sarcome de la Dure-mère ayant déterminé la Production d'une Tumeur osseuse sur la Région correspondante de la Face externe du Frontal", *Comptes rend. Soc. de Biol.*, 1887, iv, 220.
GLOBUS, J. H., "Meningiomas: Origin, Divergence in Structure and Relationship to Contiguous Tissues in Light of Phylogenesis and Ontogenesis of the Meninges, with Suggestion of a Simplified Classification of Meningeal Neoplasms", *Arch. of Neurol. and Psychiat.*, 1937, xxxviii, No. 4, 667.
GOODING, H., and STEWART, D., "A Comparative Study of Histological Preparations of Bone, which have been Treated with Different Combinations of Fixatives and Decalcifying Fluids", *Lab. Jour.*, 1932, March.
GOODING, H., and STEWART, D., "Further Investigations in the Study of Fixation and Decalcification of Bone", *Ibid.*, 1934, March.
GREIG, D. M., "On Intracranial Osteophytes", *Edin. Med. Jour.*, 1928, N.S. xxxv, 165, 237.
HEUER, G. J., and DANDY, W. E., "A Report of Seventy Cases of Brain Tumour", *Bull. Johns Hopkins Hosp.*, 1916, xxvii, 224.
HEUER, G. J., and DANDY, W. E., "Roentgenography in the Localization of Brain Tumour", *Ibid.*, 311.
JEFFERSON, G., and STEWART, D., "On the Veins of the Diploe", *Brit. Jour. Surg.*, 1928–29, xvi, 70.
KOLODNY, ANATOLE, "Cranial Changes Associated with Meningioma (Dural Endothelioma)", *Surg. Gynecol. and Obst.*, 1929, xlviii, Feb., 231.
LEBERT, H., *Traité d'Anatomie pathologique générale et spéciale*, ii, 101, *Figs.* 4–14.
LERICHE, R., and POLICARD, A., *The Normal and Pathological Physiology of Bone: Its Problems*, trans. S. MOORE and J. A. KEY, 1928. St. Louis: C. V. Mosby Co.
MALLORY, F., "The Type Cell of the So-called Dural Endothelioma", *Jour. of Med. Research*, 1920, xli, 349.
DE MARTEL, CHATELIN, and GUILLAUME, "Méningiome en Plaques de la Corticalité", *Societé de Neurologie de Paris, Séance du 22 Mai*, 1930.
MOORE, SHERWOOD, "Hyperostosis Frontalis Interna", *Surg. Gynecol. and Obst.*, 1935, lxi, Sept., 345.

PARHON and GOLDSTEIN, cited by Parhon and Nadjede.
PARHON, C., and NADJEDE, G., " Sur un nouveau Cas d'Hémicraniose ", *Revue neurol.*, 1905, xiii, 1017.
PATTERSON, NORMAN, and CAIRNS, HUGH, " Observations on the Treatment of Orbital Osteoma, with Report of a Case ", *Brit. Jour. Ophthalmol.*, 1931, xv, 458.
PENFIELD, WILDER G., " Osteogenetic Dural Endothelioma : The True Nature of Hemicraniosis ", *Jour. Neurol. and Psychopathol.*, 1923, iv, 27.
PENFIELD, WILDER G., " Cranial and Intracranial Endotheliomata Hemicraniosis ", *Surg. Gynecol. and Obst.*, 1923, xxxvi, 657.
PHEMISTER, D. B., " The Nature of Cranial Hyperostosis Overlying Endothelioma of the Meninges ", *Arch. of Surg.*, 1923, vi, March, 554.
RAND, C. W., " Osteoma of the Skull : Report of Two Cases, One being Associated with a Large Intracranial Endothelioma ", *Ibid.*, 573.
ROKITANSKY, CARL, *A Manual of Pathological Anatomy*, 1850, iii, 224. London : The Sydenham Society.
SCHMIDT, M. B., " Ueber die Pacchioni'schen Granulationen und ihr Verhältniss zu den Sarcomen und Psammomen der Dura Mater ", *Virchows Arch.*, 1903, clxx, 429.
SPILLER, W. G., " Hemicraniosis and Cure of Brain Tumor by Operation ", *Jour. Amer. Med. Assoc.*, 1907, xlix, 2059.
SOUQUES and BERTRAND, IVAN, " Examen Anatomo-pathologique d'une Vertèbre ' d'Ivoire ' dans un Cas de Cancer metastatique de la Colonne vertébrale ", *Societé de Neurologie de Paris, Séance du 2 Juillet*, 1925.
STEWART, R. M., " Localized Cranial Hyperostosis in the Insane ", *Jour. Neurol. and Psychopathol.*, 1927–28, viii, 321.
TATTERSALL, J., " A Case of Cerebral Tumour with Tumour of the Skull ", *Jour. of Ment. Sci.*, 1917, lxiii, 250.
TAYLOR, JULIAN, " Osteitis Fibrosa of Right Frontal and Sphenoid Bones ", *Proc. Roy. Soc. Med.*, 1935, xxviii, 2, 1521.
TAYLOR, JULIAN, " Invasion of the Skull by Dural Tumours ", *Brit. Jour. Surg.*, 1928, xvi, 6.
TOOTH, H. H., " Some Observations on the Growth and Survival Period of Intracranial Tumours Based on the Records of Five Hundred Cases, with Special Reference to the Pathology of the Gliomata ", *Brain*, 1912–13, xxxv, 61.
TOWNE, EDWARD BANCROFT, " Invasion of the Intracranial Venous Sinuses by Meningioma (Dural Endothelioma) ", *Ann. of Surg.*, 1926, lxxxiii, 321.
TWINING, E. W., CAIRNS, H., NORTHFIELD, D. W. C., MARTIN, J. P., JEFFERSON, G., and JUPE, M. H., " Discussion on the Value of Radiology in Neuro-surgery ", *Proc. Roy. Soc. Med.*, 1935–36, xxix, 2, 1155.
VINCENT, CL., DAVID, M., and PUECH, P., " Méningiome de la Faux du Cerveau. Ablation de la Tumeur intra et extracranienne. Sterilisation et Remise en Place du Volet osseux, Guérison ", *Soc. de Neurol. de Paris*, 1931, April.
VIRCHOW, A., *Die krankhaften Geschwülste*, 1864–65, 2, 115.
WEDL, CARL, *Rudiments of Pathological Histology*, trans. by GEORGE BUSK, F.R.S., 1855, 564. London : The Sydenham Society.
WEED, L. H., " The Cells of the Arachnoid ", *Bull. Johns Hopkins Hosp.*, 1920, xxxi, 343.
WINKELMAN, N. W., " Hyperostosis and Tumour Infiltration of the Base of the Skull associated with Overlying Meningeal Fibroblastoma ", *Arch. of Neurol. and Psychiat.*, 1930, xxiii, 494.

A LEAKING NON-SPECIFIC ANEURYSM OF THE COMMON ILIAC ARTERY, WITH EFFECTIVE TREATMENT BY WIRING

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Surgical conditions of the vascular system have been dealt with from varying angles down through the last two centuries, yet it was Vesalius in 1567 who described the first abdominal aneurysm. The literature is abundantly filled with cases of aneurysms of various areas of the body, but surprisingly scant in respect to aneurysms of the common iliac artery. One paper deals with a bilateral involvement, another with obstruction to delivery, while a third, by Zimmerman, describes a case which simulated a perinephritic abscess. The foreign literature contains several published cases in recent years.

Holcombe collected a series of 172 aneurysms over a period of ten years, and of these only 2 were confined to the common iliac. Crisp found 11 common iliac aneurysms in 552 cases of aneurysms, and stated that the order of frequency in the iliac group is: (1) Iliofemoral; (2) Femoral; (3) External iliac; (4) Common iliac; (5) Internal iliac.

THE ANATOMY OF THE PART

The case to which this report alludes is an aneurysm of the left common iliac artery, and therefore the detailed gross anatomy of this area will be recalled in order that the causes of the symptoms of the patient may be more readily comprehended.

The left common iliac artery passes obliquely laterally downwards and forwards to about the level of the sacro-iliac synchondrosis, where it bifurcates to form the internal and external iliac arteries.

Anteriorly the common iliac artery is covered with peritoneum, which closely invests this surface, the small intestine, branches of the presacral (sympathetic nerve) and parasympathetic nerves, and the superior hæmorrhoidal artery. It is crossed at its bifurcation by the left ureter and the internal spermatic vessels (ovarian).

Posteriorly, the common iliac artery rests on the 4th and 5th lumbar vertebræ and their intervening fibrocartilages, the sacrolumbar synchondrosis, the 4th lumbar sympathetic ganglion, the lumbosacral nerve trunk (L.4 and 5) (hugs the body of the 5th lumbar vertebra), the obturator nerve, the iliolumbar artery (from the posterior branch of the hypogastric—internal iliac), the sympathetic chain, and the common iliac lymph-gland.

Laterally the artery lies in close relation to the psoas muscle, covered by a firm fascial sheath, with the genitofemoral nerve on its medial border.

Medial to the common iliac artery is its corresponding vein.

Anatomically it will be recalled that all the above-mentioned structures lie in direct relationship to the triangle of Marcille, or the lumbosacral triangle, the boundaries of which are: Medial, the side of the 5th lumbar vertebra; Lateral, the inner border of the psoas muscle; Below, the upper surface of the ala of the

sacrum; Floor or posterior wall, the transverse process of the 5th lumbar vertebra and the iliolumbar ligament.

In the event of a blockage of the common iliac artery, the following anastomotic vessels would function to allow blood to pass from above to below the site of block: (1) The inferior mesenteric above anastomoses with the visceral branches of the hypogastric below; (2) The ovarian or spermatic above anastomoses with the uterine or vesical arteries below; (3) The middle sacral above, anastomoses with the lateral sacral branches of the hypogastric below.

CASE REPORT

Mr. H. N. Aged 58. White. University of Alberta Hospital, Edmonton. Admitted June 6, 1937. Operation June 21, 1937. Discharged July 5, 1937.

HISTORY.—The patient complained of: (1) Severely sharp pain in left groin of four days' duration; (2) Dull pain in left hip and down back of left leg for two and a half years; (3) Vomiting on two occasions, three days previously; (4) Frequency of urination for two days.

On June 3, while coming home from his clerking business, he was seized with a severely sharp stabbing pain in the left iliac and lumbar region, which was transmitted down the medial aspect of the thigh and into the testicle. The pain was so severe that he vomited and went into a faint. This pain persisted with less severity throughout the night, and he was unable to return to his work, having to remain in bed. He consulted the author, who advised him to be admitted to hospital. The patient, on the first visit, required two ¼-gr. injections of morphia to give relief from pain, which became more localized in the left sacro-iliac region and down the posterior aspect of the leg. For the past three years he had developed a dull pain, and at times a twinge, in the left hip area, passing into the sciatic area down to the ankle. He had obtained a belt from a local druggist which gave him some relief, and had worn this continuously for two years.

The patient has been a storekeeper for the past three years. He was formerly an insurance agent, and has worked actively most of his life. He did jumping and acrobatic feats frequently about his home. He did not give any history of direct trauma to the area of complaint. He had suffered from pleurisy in 1926, and ten years ago fell from a roof and sustained a fracture of the left tibia, which healed without incident. Subjectively, there was nothing of value in this case history.

PHYSICAL EXAMINATION.—Temperature, pulse, respiration, normal. The patient is a well-developed male, appearing younger than his stated age. Co-operates well. Pupils react to light and accommodation. Head and neck otherwise normal.

Cardiovascular System: Pulse 80, good quality. Heart normal in size. No murmurs. Vessels soft. Blood-pressure 170–90. *Lungs*: Normal. *Abdominal*: Liver, kidney, and spleen not palpable. Upon examination of the left iliac region a readily observed expansile pulsation can be seen and palpated, extending from 1½ in. of the anterior superior spine across to within 1 in. of the midline. The tumour is smooth, appearing saccular in outline, lying in the course of the iliac group of vessels. It is softer on the lateral aspect and elastic to the depressing fingers. The finger and thumb are seen to spread apart when held on this tender mass. Upon pressing on the aorta there is less expansile pulsation noted. The skin is freely movable over it. A definite bruit can be heard near the proximal end of the pulsating tumour. The comparison of the left and right femoral blood-pressures did not indicate the fairly common findings of a lower pressure in the limb involved. The left costovertebral angle is definitely tender. The dorsalis pedis artery pulsates well, but this pulse is delayed in comparison to that of the right.

A complete physical examination does not reveal anything of value to the case, other than the above.

Laboratory Findings.—Urine: Occasional red blood-cell in the first specimens, occasional trace of albumin. *Sputum*: Negative. *Blood*: R.B.C., 4,500,000; Hb, 93 per cent; W.B.C., 10,300; polymorphs 84 per cent, lymphocytes 14 per cent, eosinophils 2 per cent. Blood grouped for transfusion, if necessary. Blood Wassermann negative. Blood urea

nitrogen, 16 mg. per cent. Blood cholesterol, 198 mg. per cent. *Cerebrospinal Fluid*: Pressure 8 mm. Hg; Cells 6; Globulin negative; NaCl 0·73 per cent. Wassermann negative. Colloidal gold 010000000.

Proctoscopy.—Pulsation postero-medial to descending colon and sigmoid. Otherwise normal findings.

Cystoscopy (Dr. Gordon Ellis).—Bladder and ureteral orifices normal. Both ureters easily catheterized. Specimen negative; no shadow calculus present; left pyelogram negative; mass in left lower quadrant not in the urinary system.

Radiography (Dr. Richard Procter).—No erosion of the 4th or 5th lumbar vertebra revealed, and only a faint shadow along the common iliac area near the level of the 4th lumbar vertebra. A barium enema fails to show any abnormality in the colon.

OPERATIONS.—(1) June 12, 1937, left lumbar exploratory operation of aneurysmal sac retroperitoneally; (2) June 21, 1937, operation for insertion of silver wire into saccular aneurysm of the left common iliac artery.

Under spinal anæsthesia (Dr. C. E. Cook) and pre-operative medication with morphia $\frac{1}{6}$ gr., atropine $\frac{1}{150}$ gr., and nembutal 3 gr., a left paramedian incision was made from 2 in. above the umbilicus to 1 in. above the pubis. The abdomen was opened, the rectus being retracted laterally. Following exploration of the aorta and iliacs, the patient was placed in extreme Trendelenberg posture. The aneurysm was examined by direct vision and palpation. The sac extended from the bifurcation of the aorta to slightly beyond the bifurcation of the common iliac, a distance of 5 in., and was 3 in. in diameter. The aneurysm was adherent to the sigmoid and peritoneum, ureter, and superior mesenteric artery, by the previous effusion of blood, as was noted in the history. This effusion extended down retroperitoneally and along the psoas muscle. There was definite pulsation throughout the entire aneurysm, which gave the sensation to one's hand-grip similar to that of holding the heart of a physiological dog. There was no evidence of thrombus formation in the sac. The iliac vein was compressed on its lateral border and there was evidence of extravasated blood between the two vessels. The small bowel was packed up towards the right side and the large bowel up the left renal region. The aneurysm was then explored further by incising the peritoneum posteriorly at the sacrum and laterally, avoiding the sympathetic chain, superior mesenteric, and spermatic vessels and ureter—all of which were matted and distorted from the effusion and expanding sac. After examination of the corresponding common iliac vessels, compression of the aorta at the vertebra L.3, by means of specially devised citrate sponges on two attached forceps, was carried out by the house-surgeon, Dr. M. Hall. This resulted in control of the lower circulation and decreased pressure in the sac.

A point on the antero-lateral surface of the aneurysm was chosen for the insertion of the silver wire, and double purse-string sutures were placed through the fibrous sac, in the centre of which was passed a short curved cannula which extended into the lumen of the aneurysmal sac. Through this cannula was twisted 5 ft. of previously coiled silver wire. The end was turned bluntly, and after all was in, the double purse-string silk sutures were tied. An omental graft, folded, was placed over the insertion site and a psoas graft brought over all. As it was found that, owing to the tension of the aneurysm, the previously incised peritoneum could not be brought over the bare area, a 4-in. omental graft was stitched over the left presacral region. The toilet of the abdomen was arranged, and the wound closed layer by layer. The patient left the operating theatre in good condition. Pulse 112. Blood-pressure 90/30. A radiograph shows coils of wire in the left common iliac artery lying lateral and anterior to the 4th and part of the 5th lumbar vertebræ. (*Fig.* 437.)

The operative diagnosis was saccular aneurysm of the left common iliac artery, threatening rupture.

SUBSEQUENT PROGRESS.—The patient was kept absolutely quiet for four days in recumbency with the foot of the bed elevated, and under morphia and theobarb tablets. Slow intravenous injections of saline and glucose were carried out. He was given calcium glucinate and milk, and later an increase in high caloric diet was given. There was an uneventful recovery except for slight dilatation of the stomach with vomiting on the third post-operative day. This was quickly relieved by inserting a Wagensteen duodenal suction apparatus. The patient was discharged on the fifteenth post-operative day, and was kept at rest at home in bed for one month, following which time he progressively increased his routine life in his shop.

The patient was re-examined as follows :—

August 16, 1937 : No complaints of any kind. Is on theobarb tablets. Blood-pressure 140/80. Less pulsation. Weight 150 lb.

November 15, 1937 : Has been working regularly, no pains, eats and sleeps well. Blood-pressure 140/70. Weight 151 lb. Advised not to be too active.

December 15, 1937 : States he has never felt better. There would appear to be a definite thrombus formation in the sac, as there is not the former expansile pulsation. Blood-pressure 148/70.

FIG. 437.—Radiograph showing the silver wire in the left common iliac artery.

February 4, 1938 : Blood-pressure 138/70. No complaints of pain. A little tired from theobarb tablets. Less pulsation in the sac.

April 23, 1938 : Has been working regularly and has not had any pain since the operation. The pulsation is definitely less. Radiography at this time does not show any bony involvement. Blood-pressure 120/70. Bowels and urination good. To report in three months and continue rest intervals.

July 25, 1938 : In good health. No complaints. Working daily.

November 7, 1938 : Free from discomfort. Working at his regular business as usual.

SUMMARY

1. A leaking aneurysm of the common iliac artery, with effective treatment by inserting 5 ft. of silver wire within the sac, is presented.

2. A detailed description of the anatomy of the part involved is given.

3. The complete case history is presented, together with details of the operation and post-operative treatment. The patient is working and enjoying life at present—more than one year since the operation.

4. A roentgenographic picture showing the wire within the aneurysmal sac is included.

5. A recent bibliography of fifty references to aneurysms is given.

In conclusion, I should like to express my gratitude to the Nursing and Interne Staff of Surgery B of the University Hospital for their service, and to Nurse Calvert for her kind co-operation and attention during the period the patient was under her care; and to Dr. Richard Procter for the roentgenographic picture.

BIBLIOGRAPHY

ANDERSON, E. B., "Aneurysm Causing Sciatica", *Jour. Amer. Med. Assoc.*, 1919, xxxiii, 129.
ASPENALL, A. J., "Non-Syphilitic Aneurysm of Both Common Iliacs", *Med. Jour. of Australia*, 1934, Nov. 3, 580.
BABCOCK, W. W., "Treatment of Aneurysms", *Amer. Jour. Surg.*, 1932, xvi, 401; *Surg. Clin. N. Amer.*, 1926, vi, 38.
BELL, L. P. and TILLOTSON, R. S., *Western Jour. Surg., Obst. and Gynecol.*, 1932, xl, 475.
BOYD, L. J., "Four Thousand Cases of Aneurysms", *Amer. Jour. Med. Sci.*, 1924, clxviii, 654.
BROWN, T. K., and SOULE, S. D., "Internal Iliac Aneurysm Complicating Pregnancy", *Amer. Jour. Obst. and Gynecol.*, 1934, xxvii, May, 766.
CABOT, *New Eng. Jour. Med.*, 1937, ccxvii, Sept. 2, 403.
CALLANDER, *Ann. of Surg.*, 1920, lxxi, 428.
COKKINIS, A. J., Maingot's *Post-Graduate Surgery*, 1937.
CONYBEARE, J. J., *Textbook of Medicine*, 1932, 558.
DANDY, W. E., *Ann. of Surg.*, 1935, cii, 916.
DEAN, J. C., "Arterio-venous Aneurysm", *Wisconsin Med. Jour.*, 1934, xxxiii, 587.
DELORME, E., *Jour. Amer. Med. Assoc.*, 1921, lxxvii, 157 (abs.).
DOTT, N. M., *Edin. Med. Jour.*, 1933, Dec., 219.
ELLIOTT, *Ibid.*, 1932, Oct., 670.
ELOESSEN, L., "Aneurysm of Common Iliac Artery, Gradual Occlusion by Ligation with Free Muscle Graft", *Surg. Clin. N. Amer.*, 1923, iii, 681.
FITZGERALD, R. R., Personal communication.
GAGE, M., "Mycotic Aneurysm of Common Iliac Artery, Sympathetic Ganglion Block, etc.", *Amer. Jour. Surg.*, 1934, xxiv, 667.
GIBBONS, J. H., "The Matas Operation in the Treatment of Iliac Aneurysm", *Ann. of Surg.*, 1936, ciii, 147.
HALSTED, W. S., "Effect of Ligation of Common Iliac Artery", *Johns Hopkins Hosp. Bull.*, 1912, July.
HENNING, O., "Unusual Aneurysm of Common Iliac Artery Appearing Seven Years after Trauma", *Arch. f. klin. Chir.*, 1932, clxxi, 175.
JOACHIM, H., and GOLDZIEBER, M. A., "Bilateral Aneurysm of the Common Iliac Arteries", *Med. Press and Circ.*, 1929, cxxviii, 53.
KAMPMEIER, R. H., "Aneurysm of the Abdominal Aorta", *Amer. Jour. Med. Sci.*, 1936, July.
LA ROQUE, G. P., "Ligature of Common Iliac Artery", *Trans. Southern Surg. Assoc.*, 1931, xliii, 245.
LERICHE, "Sympathectomy for Aneurysm", *Bull. Soc. Nat. de Chir.*, 1935, lxi, 175.
LONGO, E., "Left Common Iliac Aneurysm", *Wien. klin. Woch.*, 1933, xlvi, 1168.
MCGREGOR, A. LEE, *Synopsis of Surgical Anatomy*, 1936. Bristol: John Wright & Sons.
MCGUIRE, J., *Amer. Heart Jour.*, 1934, xxxv, 360.
MACLAREN, A., "Operation for Cure of Aneurysm", *Trans. Amer. Surg. Assoc.*, 1921, xxxix, 412.
MCNEALY, Dean Lewis's *Practice of Surgery*, 1932, xii, 143.
MATAS, R., *Ann. of Surg.*, 1935, cii, 916; *Ibid.*, 1903, xxxvii, 161.
MEYERS, W., *Ibid.*, 1915, lxii, 629.
MONEZ, *Lancet*, 1933, ii, 1144.
OERTEL, *Pathology*, 25.
OSLER, *Lancet*, 1913, ii, 1248.
OTT, W. O., "Results in 21 Cases of Aneurysms", *Ann. of Surg.*, 1921, lxxiv, 513.

POWER, Sir D'ARCY, "Palliative Treatment by Wiring with Colt's Apparatus", *Brit. Jour. Surg.*, 1921, ix, 27.
REID, MONT R., "Arterio-venous Aneurysm, Effect on Heart", *Ann. of Surg.*, 1932, Jan.; "Aneurysms in Johns Hopkins Hospital up to 1932", *Arch. of Surg.*, 1926, xii, 1; "Ligation of Large Arteries", *Surg. Gynecol. and Obst.*, 1934, lviii.
RHEA, Personal communication.
ROCKEY, E. W., "Aneurysms", *Surg. Clin. N. Amer.*, 1933, xiii, 1451.
ROLLESTON, Sir H., *Encyclopædia of Medical Practice*, Vol. I.
ROMANIS, W. H. C., *Lancet*, 1935, ii, 1275; *Practitioner*, cxxxvi, 717; *The Science and Practice of Surgery*, 1935, 257.
ROSE and CARLESS'S *Manual of Surgery*, 1936, 357.
ROWLAND and TURNER, "Arteriorrhaphy", *Text-book of Surgery*, 73.
ROYSTER, H. A., *Amer. Jour. Surg.*, 1931, xiv, 79.
THOMPSON, SOUTTAR, and HOWELL, *Lancet*, 1935, i, 11.
TOWNSEND, S. R., "Aneurysm of Aorta with Rupture into Mediastinum", *Canad. Med. Assoc. Jour.*, 1936, May.
UNGER, A. S., and POPPAL, M. H., "Aneurysm of Abdominal Aorta Showing Calcification,", *Amer. Jour. Roentgenol*, 1936, xxxvi, 523.
WAKELEY, C. P. G., "Ligature of Common Iliac Artery", *Text-book of Surgical Pathology*.
WHEELER, W., "Surgery of the Blood-vessels", *Lancet*, 1933, ii, 1042.
WHITE, *Autonomic Nervous System*, 1935. London: Henry Kimpton.
WILLIAMSON, *Diseases of Children*, 1931, 182, 56.
WINSLOW, *Arch. of Surg.*, 1926, 689.
ZIMMERMAN, I. J., "Ruptured Aneurysm of Left Common Iliac Artery Simulating a Perinephritic Abscess", *New Eng. Jour. Med.*, 1938, ccxviii, Mar. 3.

VOLVULUS OF THE STOMACH ENDING IN PERFORATION

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A SMALL number of cases of volvulus of the stomach have already been recorded in the literature. The case described below is one of volvulus of the stomach ending in its perforation. This is, I believe, a very rare condition, and is possibly the only case on record.

CASE REPORT

HISTORY.—The patient, a girl of 8 years, was playing about till the evening of March 6, 1938. On returning home she did not take her food that evening, but slept well during the night. On the next morning she vomited two or three times, and suffered from nausea throughout the day. As she had no appetite, she did not take food that evening

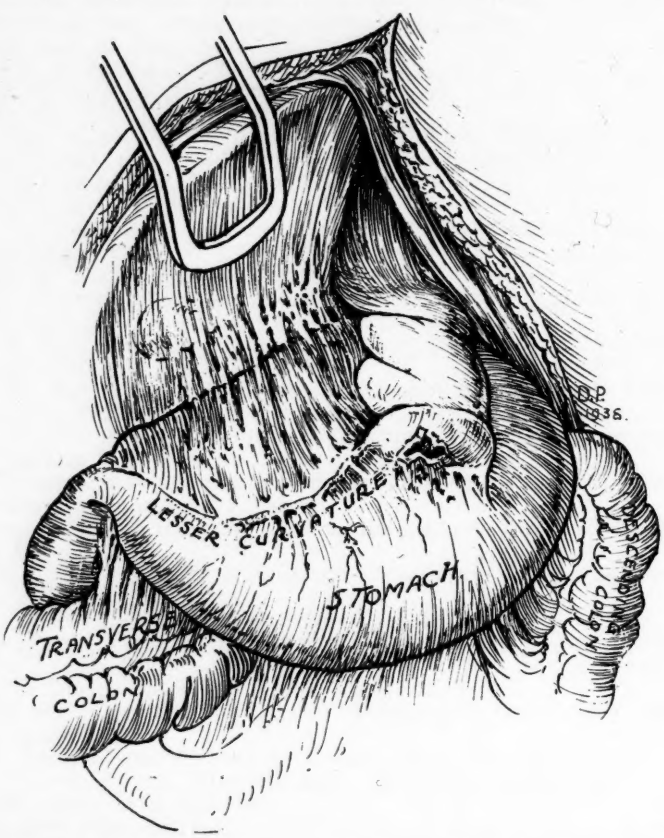

FIG. 438.—Showing the site of the perforation at the upper end of the stomach. The greater curvature has been pulled up and behind the stomach, dragging with it the transverse colon, thus masking the transverse colon from view and suggesting that it was reversely rotated.

either. She had a scanty motion. On the morning of March 8 the nausea continued, and she started vomiting everything that she took. Later on she complained of pain in the left hypochondriac region. She was therefore taken by her father to his family doctor at about 10 a.m. The doctor's notes were as follows: Abdomen soft, no tenderness or rigidity anywhere; pulse 90, tension good. She was given an alkaline and sedative mixture, and the father told to keep the girl on orange-juice and iced water. In the afternoon the girl had a temperature of 99°F. and started getting restless. She vomited whatever she took. She had nausea continually, but could not vomit unless she had taken something immediately beforehand. As she became very restless and thirsty, her father took her again to his family doctor at about 7 p.m. The doctor's findings now were as follows: Abdomen distended above the umbilicus and feels tense and tender in that area. Pulse 120, small in volume. Patient had excessive thirst, but on giving any fluid she vomited it immediately. A glycerin enema was administered, with a very poor result. The doctor now suspected this to be a case of an acute abdomen, and brought the child to my nursing home at about 9 p.m.

On Examination.—My findings were as follows: Patient very restless. Look anxious. Face pinched. Eyes sunken. Pulse 130 and small in volume. Temperature 97°. Abdomen markedly distended all over, with liver dullness completely obliterated. On palpation it was tender everywhere, and felt as if it was under great tension. In the epigastric region an indefinite swelling was felt. Its characteristics could not be defined because of abdominal distension. I therefore diagnosed the case as one of perforation of the gut, possibly complicated by dilatation of the stomach. The site of perforation I could not determine. I advised an immediate laparotomy.

At Operation.—On opening the abdomen under general anæsthesia, a large quantity of gas escaped from the abdominal cavity and dark coffee-coloured fluid was seen in the pelvis and also in the upper abdominal cavity. The appearance of the fluid suggested that it must be coming from the stomach. Accordingly the stomach was examined, and it showed a tear ¾ in. in length situated at its upper end (*Fig.* 438). The stomach was distended and blue, and the edges of the tear looked gangrenous. The tear was sutured with difficulty. Further examination revealed that it was a case of volvulus of the stomach, which had rotated on its transverse axis, causing the transverse colon to pass upwards, backwards, and a little to the left. The transverse colon was not noticeable immediately, as it was masked from view by the dilated stomach. The appearance at first suggested that the transverse colon had undergone a reverse rotation. The volvulus was corrected by me. It had caused the closure of the cardiac and pyloric ends of the stomach, which had thus become distended to an enormous size, ultimately bursting at its upper end as described. The patient's condition being very poor, the abdomen was hurriedly closed with one drainage tube in the pelvis and one in Morison's pouch.

Progress.—Vomiting continued even after the operation, and the vomit was the same dark coffee-coloured fluid which came out on the operation table. The patient was given continuous saline throughout. She died four hours after the operation.

In conclusion I have to thank Dr. Shrikhande, the family doctor, for giving me the history of the case. I also wish to thank Dr. Miss V. V. Vanikar and Dr. Gole for the help they rendered me during the operation.

VOLVULUS OF THE STOMACH ASSOCIATED WITH A RARE FORM OF DIAPHRAGMATIC HERNIA

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Volvulus of the stomach is rare. In the case recorded in this paper it was associated with an exceedingly rare form of diaphragmatic hernia. Diaphragmatic herniæ are most commonly due to a weakness in, or lack of continuity of, the phrenic tissues resulting from embryonic discrepancies, more especially at the vertebro-costal trigone. The present case is associated with hernia that does not fall into the above category. It is a hernia through the œsophageal hiatus. Such herniæ are very rare, Knaggs (1904) recording 8 cases in 63 examples of diaphragmatic hernia, whilst Keith (1910) only found 1 instance in a series of 34 specimens discovered in London Museums. Furthermore, the present specimen differs from all of the previously recorded examples.

CASE REPORT

S. C., male, Sinhalese, aged 14 months, was admitted to the Lady Ridgeway Hospital for Children, Colombo, on May 24, 1937. The father stated that the child had become very thin, and had suffered from constant vomiting for the previous three months. In the last month the father had noted that a lump appeared in the upper abdomen whenever the child took a feed. The child was very constipated, having a small dry stool about once in two days. While this history was being recorded, the child vomited, and it was noticed that the vomit contained no bile. The child was very emaciated and dehydrated. The dry, inelastic skin hung in folds over the wasted body; the anterior fontanelle was depressed, and the eyes were sunken. The pulse was feeble and thready, 134 to the minute; the respirations were 36 to the minute; and the temperature subnormal (97° F.).

A well-marked ball-like peristaltic wave could be seen every few seconds rising from under the left costal margin and passing across to the right side where it faded away. The lungs were examined, and no abnormality was detected.

The case was considered to be one of congenital hypertrophy of the pylorus, although the onset of the symptoms was much later than is usual in such cases. It was apparent that the condition of the child was desperate, but it was considered that Rammstedt's operation, rapidly performed, might give some relief.

At Operation.—The abdomen was opened by a right paramedian incision, and the greatly distended stomach was seen, covered by a sheet of peritoneum proceeding upwards from the transverse colon, which lay immediately below the stomach. When this layer of peritoneum was divided, the stomach was exposed. The pylorus was clearly seen, and on palpation it was evident that there was no hypertrophy of its musculature. The pylorus passed smoothly on to the first part of the duodenum without any kinking. Below the transverse colon lay the small intestine, which was collapsed from the duodeno-jejunal flexure downwards. At this stage the condition of the child was noted to be so bad that further operative manipulation had to be given up, and the abdomen rapidly closed. The child died shortly afterwards.

Post-mortem Examination (*Figs.* 439, 440).—On opening the abdomen by a midline incision the only structures visible on first inspection were: the stomach, which occupied the whole of the upper part of the field of view; the transverse colon, which coursed obliquely

FIG. 440.—Drawing from a photograph of the abdomino-thoracic viscera after fixation, showing the dorsal aspect. The hernial sac is visible between the thoracic surface of the diaphragm and the base of the left lung. LL., Left lung; RL., Right lung; H, Hernial sac; CC., Costal cartilages; D, Diaphragm; L.K., Left kidney; R.K., Right kidney; A, Descending aorta; OE., Œsophagus; Sp., Spleen; St., Stomach.

FIG. 439.—Drawing from a photograph of the specimen from the ventral aspect, showing the inverted position of the stomach. ST., Stomach; RL, Right lung; L, Liver; G.B., Gall-bladder; T.C. Transverse colon.

across the abdomen immediately caudal to the stomach, and coils of small intestines, which occupied the remainder of the field. The round ligament of the liver passed obliquely from the umbilicus upwards and to the right, to disappear behind the notch between the xiphoid and right costal margin. The stomach was covered with a loose layer of thin peritoneum, obviously the stretched gastrocolic omentum, for it was attached below to the transverse colon. The urachus was the only structure lying anterior to the intestines in the lower half of the belly. This occupied the normal position, but was larger than usual, and partially patent.

Disturbing the viscera revealed a small tongue of the right lobe of the liver to the right of the stomach in the right hypochondrium, the remainder of this organ being pushed backwards and to the right against the right cupola of the diaphragm. It was also observed that the stomach was much enlarged and in an inverted position, the pylorus being high up beneath the xiphoid and the greater curvature rolled forwards and upwards, with the resultant stretching out of the gastrocolic omentum already alluded to. By pulling the pylorus caudally, an orifice was found in the diaphragm, bounded in front by the left lateral ligament of the liver. The abdominal portion of the œsophagus lay to the right of the opening. The orifice itself was rounded and no abdominal organ was observed passing into it.

The left pleural cavity was next opened to ascertain the thoracic extent of the hernial diverticulum. This procedure revealed a closed, thick-walled sac, lying between the base of the left lung and the surface of the diaphragm. No abdominal protrusion into the pleural sac could be made out, and the swelling beneath the lung was tentatively assumed to be an œsophageal dilatation, or a sac connected with the stomach.

At this stage the thoracico-abdominal viscera were dissected out in one mass, together with the diaphragm, which was carefully detached all around its periphery. The mass was removed to the Anatomy Department with a view to further study after fixation and preservation. The stomach was filled by pouring in 10 per cent formalin from the œsophagus. This procedure failed to dilate the subpulmonary sac, which was therefore now considered to be a diaphragmatic hernia. The whole specimen was allowed to harden for some days in 10 per cent formalin, after which it was carefully dissected. The record of the dissection is given below.

ANATOMICAL INVESTIGATION

As the anomaly in the diaphragm is the primary anatomical feature in the present case, this structure will be dealt with first.

The Diaphragm.—Most diaphragmatic herniæ are due to weaknesses in, or lack of continuity of, the musculature of the diaphragm (Knaggs, 1904, Keith, 1910). Such anomalies are more frequent on the left side, and the commonest form is herniation through the vertebrocostal trigone. The wall of the hernial sac in these cases is the stretched-out pleuroperitoneal membrane, if this is intact; otherwise there is a free communication between the peritoneal and pleural cavities.

But in the present instance the above conditions are not found. The only circumstance which this case has in common with the above type is its left-sidedness. Developmentally the diaphragm is complete. Its vertebral and costal portions are not separated by a hiatus or trigone. The central tendon is complete and of practically normal form. It has the trefoil form, but is much elongated transversely. The caval opening is in the right leaflet, near its base, half way between the dorsal and ventral margins of the tendon. The aortic opening is also in the normal position, between the two crura, and guarded by the middle arcuate ligament. The œsophageal hiatus, on the contrary, is grossly abnormal, chiefly in size. The fibres of the right crus proceed upwards in the usual manner to form a sphincteric arrangement round the œsophageal opening, but the fibres passing to the left of the opening take a very wide sweep and become converted into a fibrous marginal strand as they do so. In this way a large orifice, far too big for the mere passage of the œsophagus, is formed. The œsophagus occupies the right margin of the opening and fills only about one-quarter of it.

The right margin of the opening is not guarded by a fibrous border like the left, but is completely fleshy. Fleshy tissue also intervenes between the dorsal margin of the opening and the aortic hiatus. The ventral (anterior) half of the margin of

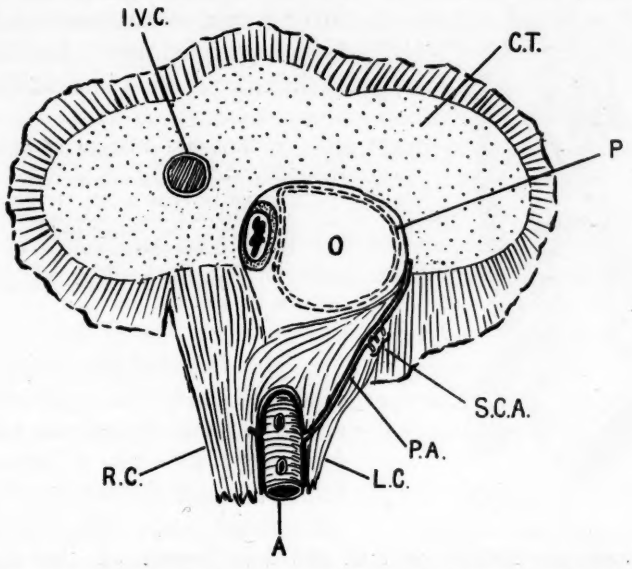

FIG. 441.—Structure of diaphragm in the present case. C.T., Central tendon; L.C., Left crus; R.C., Right crus; A, Aorta; P.A., Inferior phrenic artery; S.C.A., Superior capsular arteries; P, Peritoneum, cut edge of lining of hernial sac; O, Orifice of hernial sac; I.V.C., Inferior vena cava.

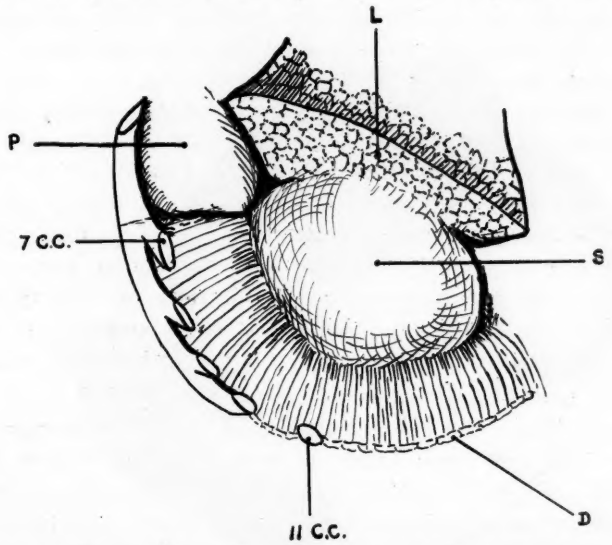

FIG. 442.—Left lateral aspect of the hernial sac and related parts from the thoracic side. 7 C.C., 7th costal cartilage; P, Pericardium; L, Left lung, basal surface turned upwards; S, Sac, covered by pleura, which is seen passing on to the diaphragm below and to the basal surface of the lung above; D, Diaphragm; II C.C., 11th costal cartilage.

the opening is bounded by central tendon. The left inferior phrenic artery ascends on the left crus and encircles the margin of the œsophageal orifice, lying actually on the fibrous band. The branches of this artery to the left suprarenal gland are given

off downwards and laterally, for this gland lies lateral to the hernial opening, and not medial to it as in the case of the commoner type of diaphragmatic hernia (*vide* Frazer, 1937). The opening measures 25 mm. across and 23 mm. dorsoventrally. The œsophagus fills 6·5 mm. of the transverse space, the rest being occupied by a hernial protrusion of peritoneum passing into the thorax (*Fig.* 441).

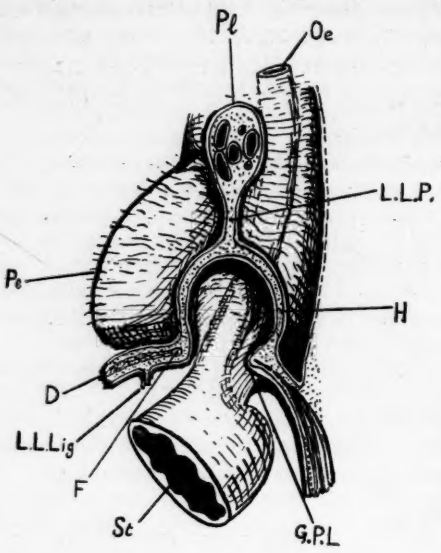

FIG. 443.—Diagram of a sagittal section through the hernial sac, viewed from the left, to show its relation to the mediastinum and the ligamentum latum pulmonis. Pl, Pleura surrounding hilum of left lung; L.L.P., Ligamentum latum pulmonis; Oe, Œsophagus; H, Hernial sac; G.P.L., Gastrophrenic ligament; St, Stomach; F, Fibrous ring; L.L.Lig, Left lateral ligament of the liver; D, Diaphragmatic musculature; Pe, Peritoneum.

The Hernial Sac (*Figs.* 442, 443).—As explained above, this is primarily a protrusion of peritoneum through an enlarged œsophageal hiatus into the thorax. Seen from the thoracic aspect the sac is subglobular, with a rather thick wall, lying between the base of the left lung and the medial part of the left cupola of the diaphragm. The lung is partly adherent to it, whilst to the medial side and anteriorly lies the

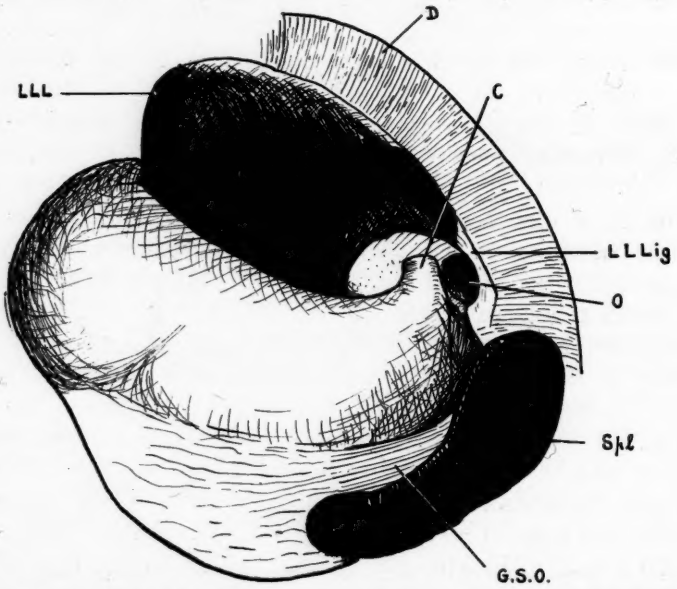

FIG. 444.—View from the abdominal aspect of the diaphragm, to show the orifice of the hernial sac and its relations. The stomach has been depressed and the spleen pulled aside. D, Diaphragm, abdominal surface; LLL, Left lobe of the liver; LLLig, Left lateral ligament of the liver; O, Orifice of the sac; C, Cardiac end of the stomach; Spl, Spleen; G.S.O., Gastrosplenic omentum.

pericardium, from the posterior margin of which the pleura is sharply reflected on to the front of the sac. Posteriorly the sac extends into the pulmonary groove

of the thoracic wall. It is clear from its position that the sac is formed by the separation of the layers of the ligamentum latum pulmonis by the encroachment of the peritoneal protrusion from below. The wall of the sac, therefore, consists of a lining of peritoneum, an outer covering of pleura, and a thick layer of loose mesodermal tissue between them. There is no communication between pleural and peritoneal cavities. No previously recorded case of hernia through the œsophageal orifice had a sac like the one just described (Knaggs, 1904).

During the passage of peritoneum from the abdomen into the thorax, the gastrophrenic fold or ligament (i.e., dorsal mesogastrium) has been dragged upwards on to the posterior wall of the sac. This was the only structure seen in the sac both at operation and post mortem. Œdema of the extraperitoneal tissue has resulted in a thickening of this fold in the same manner as the thickening of the wall of the sac itself. Its presence suggests a gradual sliding of the peritoneum from abdomen to thorax.

The external dimensions of the sac are as follows: anteroposterior 45 mm., supero-inferior 38 mm., transverse 40 mm. The orifice of the sac, as seen from the abdominal aspect, prior to the disturbance of the peritoneum, has been sufficiently described already (*Fig.* 444). Its internal dimensions were 16 mm. across and 11 mm. dorsoventrally.

The Alimentary System.—Several instances of diaphragmatic hernia are recorded in which, quite apart from the fact that parts of the alimentary tube may be found within the hernial sac, there are anomalies of arrangement (e.g., Sagal, 1933; Elward, 1936). Amongst the anomalies recorded are diverticulum of the œsophagus, herniation of the cardiac part of the stomach through the œsophageal opening, and inversion of the stomach. In the present case the œsophagus is more capacious than in the normal child, especially at its lower end, but there is no diverticulum. There is no constriction at the level of passage through the diaphragm; in fact, the cardio-œsophageal junction is definitely on the thoracic side of the diaphragm. The abdominal part of the œsophagus cannot therefore be said to be present.

The stomach, as already remarked, is 'inverted'. This term is not quite an adequate one. The change in position of the stomach involves a departure from the normal in respect of two planes of space. The cardia is the most fixed point. The body of the stomach appears to move forwards and then its pyloric end to move upwards, so as to bring it into contact with the diaphragm, and also for it to lie almost directly anterior to the cardia. The lesser curvature is, therefore, very short and sharply bent. At the same time the greater curvature is rolled forwards and then upwards, dragging the gastrocolic omentum with it, so that the latter becomes stretched out to form a thin sheet over the front of the stomach. This anomalous position falls into the category of 'partial volvulus'. The liver, meanwhile, travels backwards and to the right, the left lobe becoming grossly compressed by the rotating and enlarging stomach. Whether the rotation or the enlargement of the stomach is the primary feature is difficult to decide. The enlargement is undoubtedly due to interference with the emptying mechanism.

The inversion of the stomach has had its effect on the duodenum. Instead of this exhibiting a C-shaped form, it takes the course of a J, with the small limb to the left. The long limb descends vertically from the pylorus for 85 mm. and then turns sharply to the left and upwards for 35 mm. The duodenojejunal flexure is in

the normal position and has the usual relation to the root of the mesentery and the vessels contained therein. The remainder of the small intestine calls for no special remark beyond what has been stated already in the case report. The large intestine, however, presents some noteworthy features, chiefly of a primitive nature. The cæcum is preternaturally mobile and has its long axis obliquely placed. It has just begun to assume the asymmetrical form. The appendix is very long, much coiled, and has a mesentery almost to its tip. The ascending colon is very short, proceeds obliquely upwards and to the left, and becomes continuous with the transverse colon with scarcely any hepatic flexure (infantile condition). It is provided with a short mesentery, which is longer in the middle than at its two extremities. The transverse colon is almost straight, crossing the abdomen with a slight upward trend at its left extremity. There is a sharp splenic flexure, followed by a long descending colon which is provided with a complete mesocolon. The rest of the gut shows no abnormality. There is no distension of the sigmoid colon as in the case reported by Barclay Smith (1898).

VOLVULUS OF THE STOMACH

As this is the most important aspect of the case from the clinical point of view, some further discussion thereon is necessary. Volvulus of the stomach was first observed by Berti (1866) at a post-mortem on a patient in whom the volvulus complicated a tumour of the stomach wall. There were only 70 cases on record of the complete variety of volvulus in 1935 (Weeder).

Mechanics of Volvulus of the Stomach.—The stomach is normally fixed by the œsophagus to the œsophageal hiatus of the diaphragm, the attachment being re-inforced by the gastro-phrenic ligament. For the rest, the stomach lies comparatively free, suspended only by folds of peritoneum. The pylorus is, however, a relatively fixed point on account of the retroperitoneal position of the duodenum. From this, it follows that a complete volvulus of the stomach will occur in relation to a line joining the two fixed points, cardia and pylorus. By complete volvulus is meant the condition in which the whole viscus is affected, whereas a partial volvulus affects a portion of the stomach only. There are two types of complete volvulus; one comparable to a volvulus of the sigmoid colon, in which the viscus pivots round a taut mesentery; and the other comparable to a log of wood floating in water and rolling on its own long axis. In the first type the taut mesentery is the lesser omentum, and the pylorus, which is more mobile than usual in these cases, travels to the left in front of the fundus, twisting the stomach on an axis which is at right angles to the cardiopyloric line. In the second type the stomach rolls on its own long axis, which is a line passing from cardia to pylorus through the centre of the viscus.

Von Haberer (quoted by Buchanan) suggested the cumbrous but expressive terms ' volvulus mesenterio-axialis ' for the first type, and ' volvulus organo-axialis ' for the second type. Many writers have used indiscriminately the terms volvulus and torsion, but Becker (1927) differentiates between volvulus (*Magenverschlungung*) and torsion (*Magenverdrehung*), an accurate distinction, for ' torsion ' is derived from the Latin *torqueo*, meaning ' I twist ', and ' volvulus ' from the Latin *volvo*, meaning ' I roll ', and in the first type the stomach twists on its taut mesentery, while in the second type the stomach rolls on its own long axis. The term ' volvulus ' is, however, used generally to include all the varieties of twisting and rolling of the stomach, and we have therefore retained von Haberer's nomenclature.

Volvulus Mesenterio-axialis.—In this type, a deep sulcus is produced in the lesser curve, as the pylorus travels over to the left, the sulcus separating the cardiac and pyloric parts of the stomach. The pyloric pouch becomes enormously distended on account of the occlusion of the pylorus, produced by the twisting and kinking resulting from its change of position. The distended pyloric pouch now crosses the fundus and lies with its posterior surface facing forwards. In the

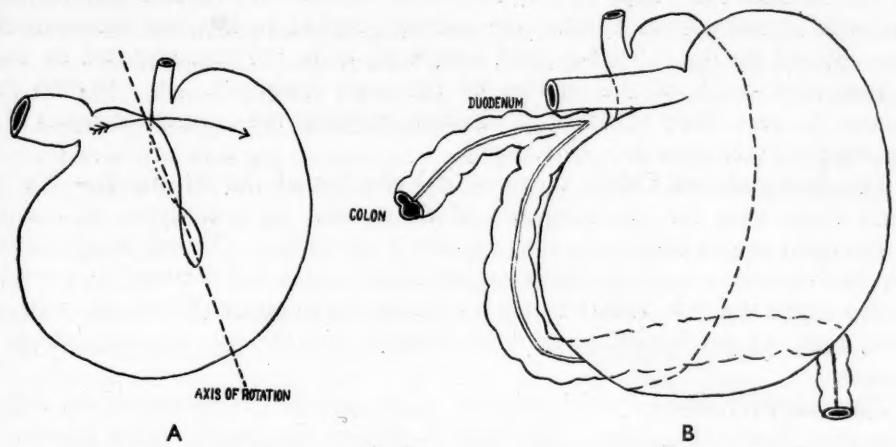

FIG. 445.—Two stages in the formation of Type I volvulus of the stomach—volvulus mesenterio-axialis. A, Early stage. Arrow indicates direction of movement dotted line axis of rotation. B, Final stage (modified after Buchanan)

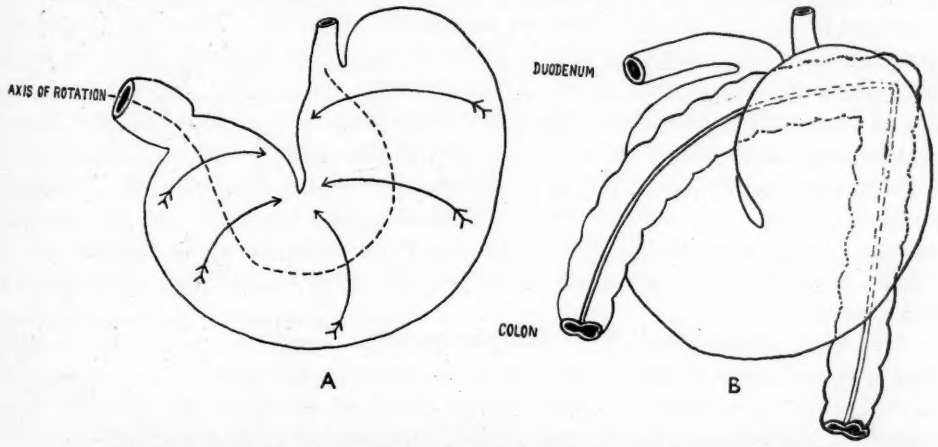

FIG. 446.—Two stages in the formation of Type II volvulus of the stomach—volvulus organo-axialis. A, Early stage. Arrows indicate direction of rotation; dotted line axis of rotation. B, Final stage (modified after Thorek). The relation of the colon is different from that found in the present case.

more complete grades of torsion, even the cardiac orifice of the stomach becomes occluded. (*Fig.* 445.)

The right half of the colon is carried with the pylorus upwards and to the left, from where it descends sharply over the distended pyloric pouch to below and behind it, reappearing again on the left side from the splenic flexure. The sharp kink in the colon produced by this displacement leads sometimes to great distension of the right half of the colon.

Volvulus Organo-axialis.—In this type, as the stomach rolls on its own long axis, the relatively mobile pylorus is drawn towards the fixed cardia and a deep V sulcus appears on the lesser curve. As the stomach rotates upwards from below, the greater curve comes to lie below the liver, while the lesser curve rotates to the position of the greater curve and is liable to be mistaken for this curve. The transverse colon usually travels upwards with the great curve of the stomach, in which case the stomach lies veiled by the transverse mesocolon. Occasionally the colon remains in its normal place below the stomach, and in such a case the stomach is either covered by the expanded great omentum, as in the case reported by us, or lies bare as a result of the rupture of the great omentum. A stretched great omentum is very likely to occur in children in whom the component layers of the membrane are still separate. (*Fig.* 446.)

Displacement of Other Organs in Volvulus of the Stomach.—We have already dealt with the displacements of the colon. In both types of complete volvulus other organs besides the stomach may be disturbed. Thorek (1923) collected from the literature 4 cases in which torsion of the spleen had occurred as a result of volvulus of the stomach, and there are 2 cases in the literature (Wiesinger, 1901, and Hailes, 1923) of patches of fat necrosis showing that the traction had injured the pancreas.

Partial Volvulus.—Partial volvulus of the stomach is always of the organo-axialis type (Buchanan, 1930). The pyloric part of the greater curve rotates to a greater extent than the cardiac part, and the occlusion of the pylorus produced by the torsion leads to moderate distension of the pyloric pouch of the stomach, which is separated from the cardiac pouch by a V sulcus on the lesser curve. The angle of rotation in partial volvulus does not exceed 180° as a rule. Morrison (1931) described an interesting case of partial volvulus of the stomach which differs in several points from the description given in the previous paragraph. In this case four-fifths of the stomach from the cardiac orifice was twisted on its own long axis to such a degree as to cause strangulation of this part of the stomach from interference with its blood-supply. Partial volvulus is always associated with gastric or extragastric lesions (Buchanan), the commonest being an hour-glass stomach. In an hour-glass stomach, the pyloric pouch rotates on its two extremities. A tumour of the stomach wall or an inflammatory lesion of the stomach wall may also initiate a partial volvulus.

Volvulus Associated with Diaphragmatic Lesions.—There is an interesting group of cases of partial volvulus of the stomach associated with lesions of the left half of the diaphragm. Payer (1907) found 12 instances of volvulus of the stomach in the records of 500 cases of diaphragmatic herniæ, and German writers have drawn attention to its association with constitutional weakness of the left half of the diaphragm. The condition has also been recorded following paralysis of the left half of the diaphragm from the operation of avulsion of the phrenic nerve (Bérard, Delore, and Bonafé, 1932; Bonafé and Poulain, 1932).

It has been suggested that in diaphragmatic hernia there is a lengthening of the mesenteries of the stomach, as a result of the stomach being drawn up into the hernial sac, and that the same thing occurs with the raised cupola of a paralysed diaphragm. There is, however, another factor which must be considered, especially as in many of these cases the stomach may never have occupied the hernial sac. Any defect in the left cupola of the diaphragm must diminish the downward thrust of the

diaphragm on the stomach. It would appear to us that there is normally a balance between two opposing forces acting on the stomach; namely, the downward thrust of the diaphragm and the upward thrust of the transverse colon and mesocolon, from the pressure on them of the abdominal wall. Should the balance be upset by diminution of the downward thrust of the diaphragm, the upward thrust of the colon will rotate the greater curvature upwards and forwards, and thus initiate a volvulus of the organo-axialis type.

Other Factors which may Favour the Occurrence of Volvulus.—Volvulus would occur more easily in cases in which the mesenteries of the stomach are elongated, and the stomach ptosed. Rupture of the peritoneal folds of the stomach would also predispose to the occurrence of volvulus.

In children the stomach is normally horseshoe-shaped, with the pylorus high above the greater curve, a condition of relative gastroptosis, and Simmonds (1907) points out that volvulus is for this reason more likely to occur in children. On the other hand, in Pendl's (1904) case, the gastrocolic ligament was by contrast thickened and sclerosed, and he believes that the thickened ligament permitted the colon to pull on the greater curve and thus start a volvulus. French writers believe that a colon distended with gas pushes up the greater curve of the stomach and places it in a position favourable for the occurrence of a volvulus. Payer (1909) drew attention to the fact that in normal gastric peristalsis, as observed after a barium meal on the X-ray screen, the greater curve rotates upwards and forwards in the same direction that it does in the organo-axialis type of volvulus, and Payer suggested the name 'iso-peristaltic' volvulus for this type. It would appear as if an exaggeration of normal peristalsis in an overdistended stomach would lead to volvulus.

Whatever the predisposing causes, there is no doubt that the exciting cause in many of the recorded cases is overfilling of the stomach. Kerr (1912) suggested that acute dilatation occurs in these cases from loss of splanchnic control. Once the volvulus has occurred, reduction is prevented by the pressure of the anterior abdominal wall, which holds the distended stomach compressed between the abdominal wall and the diaphragm (Thorek).

The Surgical Treatment of Volvulus of the Stomach.—Operation in cases of total volvulus has given 60 per cent of recoveries, and in the case of partial volvulus operation is benignant (Thorek). In many of the cases of total volvulus unsuccessfully operated on, death was due to cardiac failure from the embarrassment of the heart caused by the gross distension of the upper abdomen. In many of the cases of total volvulus, it is necessary to deflate the grossly distended stomach by puncturing it with a trocar, before the volvulus can be undone. The site of the trooar puncture should be closed by suture before the volvulus is untwisted, as the puncture may subsequently recede to an inaccessible position. The ease with which a volvulus is untwisted, once it has been punctured with a trocar, led Berg (1897) to suggest that cases of idiopathic rupture of the stomach may in reality have been cases of volvulus in which spontaneous rupture of the overdistended viscus had led to reduction of the volvulus.

In the severely ill, it may suffice to untwist the volvulus only. In others something must be done to prevent a recurrence. Thorek did a gastropexy by the Rovsing method, a technique well suited for this type of case. Niosi performed a gastro-enterostomy in order to fix the stomach and to prevent over-filling.

DISCUSSION

The case reported is one of partial volvulus of the stomach of the organo-axialis type. As the clinical symptoms and signs have shown, the volvulus had led to chronic pyloric obstruction, and death was due to the starvation consequent on this. The volvulus is obviously secondary to the diaphragmatic anomaly, which is a rare type of congenital hernia that did not cause the death of the child at birth by interference with the respiratory mechanism.

An apparently puzzling feature of the case is the absence of any kink or twist in pylorus or duodenum when the parts were inspected at operation and later at the more detailed examination made after death. It is probable that the relief of tension produced by the abdominal incision sufficed to open out the kink in the duodenum or pylorus, even though it still left the stomach in the condition of volvulus. The case is further unusual in that the volvulus had been present for some months, and had produced the symptoms of a chronic pyloric obstruction.

BIBLIOGRAPHY

BARCLAY-SMITH, E., *Jour. of Anat.*, 1898, xxxii, 341.
BECKER, *Arch. f. klin. Chir.*, 1927, cxl, 397.
BÉRARD, DELORE, and BONAFÉ, *Lyon chir.*, 1932, xxix, 463.
BERG, *Nord. Med. Ark.*, 1897, viii, No. 19 (Stockholm).
BERTI, *Gaz. Med. Ital., prov. Venete*, 1866, ix, 139 (Padua).
BONAFÉ and POULAIN, *Presse méd.*, 1932, xl, 1104.
BUCHANAN, J., *Brit. Jour. Surg.*, 1930, xviii, 99.
ELWARD, J. F., *Amer. Jour. Roentgenol.*, 1936, xxxvi, 305.
FRAZER, J. E. S., *Buchanan's Manual of Anatomy*, 1937, 6th ed., 838.
HABERER, VON, quoted by Buchanan.
HAILES, *Med. Jour. of Australia*, 1923, No. 544.
KEITH, A., *Brit. Med. Jour.*, 1910, ii, 1297.
KERR, H. H., *Ann. of Surg.*, 1912, lvi, 697.
KNAGGS, R. LAWFORD, *Lancet*, 1904, ii, 358.
MORRISON, W. A., *Surg. Gynecol. and Obst.*, 1931, lii, 871.
PENDL, L., *Wien. klin. Woch.*, 1904, No. 70.
PAYER, A., *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1909, xx, 44.
SAGAL, Z., *Amer. Jour. Roentgenol.*, 1933, xxx, 206.
SIMMONDS (Jena, G. Fischer), 1907, quoted by Kerr.
THOREK, M., *Jour. Amer. Med. Assoc.*, 1923, xxxi, 636.
WEEDER, S. D., *Ann. of Surg.*, 1935, cii, 382.
WIESINGER, D., *Deut. med. Woch.*, 1901, 83.

SHORT NOTES OF RARE OR OBSCURE CASES

ADENOMA OF THE STOMACH

By A. LATIMER WALKER
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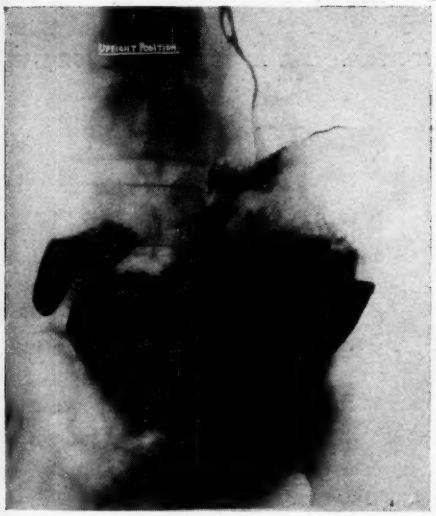
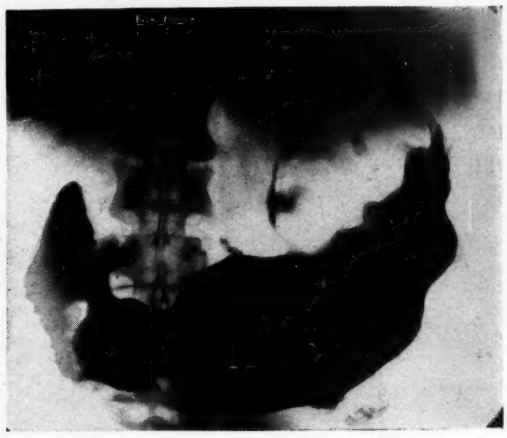
THE following case is thought worthy of record owing to the infrequency of the condition :—

HISTORY.—Mrs. B., a housewife aged 57, was sent to the medical out-patient department of the Huddersfield Royal Infirmary in May, 1937, with a long history of anæmia. Seven years previously her uterus, ovaries, and appendix had been removed, otherwise there was nothing noteworthy in her previous medical history.

ON EXAMINATION.—She was very anæmic, the pulse was of poor volume and tension, and the heart-sounds weak. The blood-count showed a severe secondary anæmia, as follows: Hb 30 per cent; R.B.C. 3,190,000; W.B.C. 9,400—





FIGS. 447, 448.—Radiographs taken following a barium meal, and showing a very large filling defect in the upper portion of the stomach.

polymorphs 88 per cent, lymphocytes 12 per cent. Colour index 0·48. On abdominal examination a hard mobile tumour was felt in the left epigastric area. A barium meal examination showed a very large filling defect in the upper limb of the stomach (*Figs.* 447, 448).

She was referred to me on June 9, 1937, when I found a hard mobile tumour about the size of an apple above and to the left of the umbilicus. The patient stated that she had felt this for five years. A fractional test-meal examination showed achlorhydria and no blood.

ON ADMISSION TO SURGICAL WARD.—She was admitted to the surgical side on June 21, and a blood-count was as follows: Hb 24 per cent; R.B.C. 2,690,000; W.B.C. 9,400—neutrophil polymorphs 87 per cent, lymphocytes 9 per cent, transitionals 4 per cent. Colour index 0·45. The red cells showed very marked loss of hæmoglobin and changes in shape. A few microcytes were present.

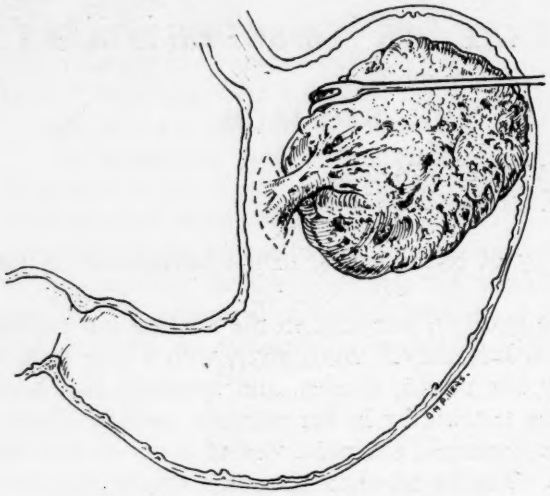


FIG. 449.—Diagram of stomach in frontal section, showing the size and position of the adenoma.

On June 25 a blood transfusion was given.

On June 28 the R.B.C. were 2,030,000 and the Hb 30 per cent.

AT OPERATION.—On July 2, under general anæsthesia, a paramedial incision was made to the left of the middle line from the ensiform to below the umbilicus. The mass was found to be inside the stomach on the posterior wall, and the size of a medium-sized apple (*Fig.* 449). The liver and spleen appeared normal, and no enlarged glands were felt. An incision 4 in. long was

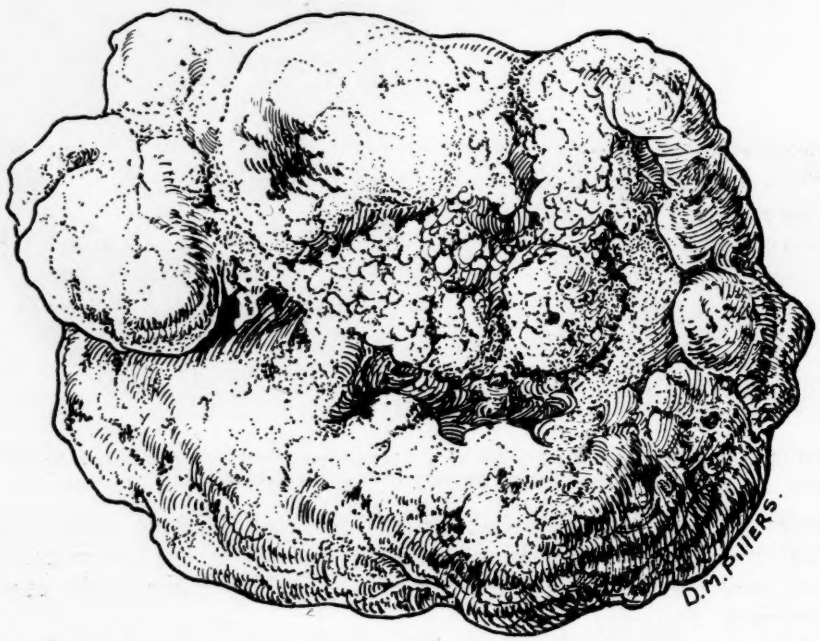


FIG. 450.—Drawing of the posterior view of the fibro-adenoma after removal. (*Actual size.*)

made in the front wall of the stomach, and the tumour was found to have a pedicle 1 by ½ in. situated on the posterior wall, just below the lesser curvature

and towards the cardiac end. The tumour and its pedicle were removed, together with a small piece of the posterior wall of the stomach from which it arose, the lesser sac being opened. The wound in the posterior wall of the stomach was sutured in two layers and the anterior stomach wound similarly closed. The wound in the abdominal wall was closed in the usual way. At the end of the operation 15 oz. of blood were given intravenously.

FIG. 451.—Photomicrograph of section of the tumour

SUBSEQUENT PROGRESS.—After the operation she made an uninterrupted recovery, and is at present very well. She is taking hydrochloric acid for the achlorhydria.

PATHOLOGICAL REPORT.—The mass is a firm, hard, nodular tumour 9.5 cm. long, 7·0 cm. wide, and 5·5 cm. thick (*Fig.* 450). The weight is 200 g. There is no sign of ulceration. On section the tumour is a simple adenoma of the stomach (*Fig.* 451).

COMMENT

Incidence of Benign Tumours of the Stomach.—There seems to be considerable difference between the surgical and pathological estimates of the relative frequency of malignant and benign tumours of the stomach. Hunt[1] gives the incidence of benign tumours from the surgical side as from 0·5 to 5·0 per cent of all gastric tumours; whereas from post-mortem examinations they were 26 per cent of gastric tumours.

Adenomata appear to be among the less frequent types of benign stomach tumours. Minnes and Geschickter[2] have reviewed all the recorded cases of benign tumours of the stomach, and of these 4·5 per cent (42 out of 930) were adenomata.

These benign tumours occur in all parts of the stomach.

Symptoms.—Many benign tumours are symptomless and have been discovered post mortem. Anæmia is a frequent symptom, and may be associated with hæmatemesis or occult bleeding. Occasionally severe or even fatal hæmorrhage has occurred. Dyspepsia or ulcer symptoms may occur. A gastric or duodenal ulcer may be present. and in some cases the ulcer has been situated over the tumour. A pedunculated tumour, or one near the pylorus, may cause symptoms of pyloric obstruction. Achlorhydria has been reported, and also hyperchlorhydria. This probably depends on the condition of the remainder of the stomach.

X-ray Findings.—Moore (quoted by Hunt[1]) states that the characteristics of benign stomach tumour are: (1) The filling defect is generally central rather than marginal, and is usually round or oval and smoothly and sharply contoured; (2) The outline of the gastric shadow is usually preserved; (3) Absence of spasm; 4) Uninterrupted peristalsis.

Carter and Laing[3] state that most benign tumours show circumscribed filling defects on the gastric walls, leaving the curvatures regular and free for peristaltic movemets; they do not show a niche, incisura, or other evidence of spasm; and gastric retention is rare except when the lesion is near or at the pylorus.

Treatment.—Starr Judd and Hoerner[4] advise that all benign tumours should be removed on account of the risk of malignant transformation. In an analysis of 50 cases the following operations were done: local excision, 20; local excision and gastrojejunostomy, 4; Billroth I, 5; Billroth II, 8; anterior or posterior Polya, 11; sleeve resection, 2.

Summary.—A case of simple adenoma of the stomach, successfully removed by transgastric resection is reported, together with a short résumé of the incidence, symptoms, and treatment of the condition.

I am indebted to Dr. J. W. Hirst and Dr. McCully for notes on the case; to Dr. Denton Guest for the pathological investigations; and to Dr. Raffan for the X-ray reports.

REFERENCES

[1] HUNT, V. C., *Surgery*, 1937, i, 711.
[2] MINNES, J. F., and GESCHICKTER, C. F., *Amer. Jour. Cancer*, 1936, Sept., 136.
[3] CARTER, R. A., and LAING, D. R., *Radiology*, 1937, xxviii, March, 301.
[4] JUDD, E. STARR, and HOERNER, M. T., *Amer. Jour. Surg.*, 1936, xxxi, March, 427.

A CASE OF TRAUMATIC SEVERANCE OF THE COMMON BILE-DUCT

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AN uncomplicated case of rupture of the common bile-duct due to injury is an unusual occurrence, and the following case in which this occurred is therefore recorded, together with some remarks regarding the aetiology and treatment.

HISTORY.—P. C., aged 32, a steel erector, whilst following his employment on April 5, 1938, was struck in the back by a steel rafter (weight about 3 cwt.) which was swinging on a crane, and was thrown forward on to his stomach against a pile of steel girders. Although he was hit in the back he " knew at once it was

his stomach that was injured", owing to the sudden excrutiating abdominal pain experienced. He was able to walk to the ambulance, and after some attention to his spine, was brought and admitted to the Cardiff Royal Infirmary

ON ADMISSION.—The patient vomited first on arrival at the hospital, about an hour after the accident, the vomit consisting of undigested food, as he had just eaten his dinner. After this, he vomited quantities of food continuously. He was obviously suffering from a severe degree of peritonism—he was in very severe abdominal pain; the legs were flexed, and there was board-like rigidity of the abdominal muscles. There was some bruising of the lower part of the back, but none of the abdomen. The abdomen was exquisitely tender all over, but the greatest tenderness was elicited in the right hypochondriac region. There was no dullness in the flanks, and liver dullness was present. Temperature 96°, pulse 90, respirations 18. Provisional diagnosis: Ruptured viscus, probably small intestine.

AT OPERATION.—Under spinal anæsthesia (12 c.c. 1–1500 percaine), supplemented by gas and oxygen, the abdomen was opened by a long right paramedian incision. On opening the peritoneum, the peritoneal cavity was found to be full of bile-stained fluid; the whole length of the small intestine was rapidly examined, but no injury was found. The incision was then enlarged upwards in order to examine the biliary organs, and a small tear at the first and second parts of the duodenum was found. Morison's pouch was found to be full of bile, which re-accumulated at once after emptying. In order to expose the injury to the duodenum, the right rectus was divided and the rent in the duodenum repaired. It was considered at the time that the escape of bile came from the tear in the duodenum. The bile in Morison's pouch was then emptied and preparations made for closure of the abdomen. Fortunately, however, before closing the abdomen a large volume of bile welled up and refilled Morison's pouch, and it was thus determined that the escape of bile was from elsewhere than the damaged duodenum.

The biliary organs were then carefully examined. The gall-bladder was normal and distended, and upon applying pressure to it, bile was found to exude from a minute opening in the peritoneum in the free border of the foramen of Winslow; this opening was enlarged by gentle gauze dissection, and it was found that the common bile-duct was completely severed. It was not possible to find the lower end of the duct, as it had apparently retracted behind the duodenum. As far as could be ascertained, there was no damage to the portal vein or hepatic artery, as there was no blood in the exudate.

Owing to the retraction of the lower end of the common bile-duct, direct repair by anastomosis was not possible. The following two alternatives presented themselves: (1) Direct anastomosis of the upper end of the common bile-duct with the stomach, duodenum, or small intestine; (2) Closure of the upper end of the common bile-duct and anastomosis of the gall-bladder with the stomach or duodenum. The second method was decided upon, as it was considered the simpler method. The torn lower end of the upper segment of the common bile-duct was closed and a cholecystgastrostomy performed. The abdomen was closed with drainage in the usual manner.

SUBSEQUENT PROGRESS.—The patient made an uninterrupted recovery; and no jaundice was noticed at any time during his convalescence. He was perfectly well two months later, and was back at work.

Comments.—The case presented the following interesting features:—

1. The mechanism by which the trauma was applied so that the common bile-duct alone was injured. The other structures in the free margin of the foramen of Winslow were spared (hepatic artery and portal vein). Of these three structures one might have reasonably supposed that the portal vein was the one structure most likely to sustain damage, this being the frailest structure and the most inelastic. I do not know of any reasonable explanation to account for the bile-duct being torn.

2. There was never at any time any jaundice after the operation. With the common bile-duct completely closed, it would be reasonable to suppose that some bile might have become absorbed into the blood-stream; as, although there was an anastomosis between the gall-bladder and the stomach which would efficiently drain the former, the presence of the valve of Heister, one would have thought, would have tended, at any rate at first, to prevent a perfectly free flow of bile from the hepatic ducts into the gall-bladder.

The only experience that the writer has had of the operation of cholecystogastrostomy has been for obstruction due to carcinoma of the head of the pancreas, and he has not been impressed with the efficacy of the operation in relieving jaundice in this condition.

ACUTE INTESTINAL OBSTRUCTION IN A BABY 15 MONTHS OLD
HERNIA THROUGH THE FORAMEN OF WINSLOW

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History.—On June 3, 1938, a boy aged 15 months was admitted to hospital in a collapsed condition and obviously in great pain at two-minute intervals. There was a history of gastro-enteritis the previous week.

On Examination.—Clinically, the picture presented was that of intussusception, and blood and mucus were passed immediately after admission.

The abdomen was soft, but palpation of the right iliac fossa was resented. No 'sausage' tumour could be felt, but there was a curious indefinite 'lump' high up in the epigastrium.

At Operation.—An immediate laparotomy was performed, and after careful exploration I was amazed to find the tip of the appendix in the neighbourhood of the pylorus. The cæcum, the appendix, and nine inches of the ileum had passed through the foramen of Winslow, and lay behind the stomach to form the indefinite 'lump'. After reduction of the hernia, a slight thrombosis of the ileum was observed.

Before closing the abdomen I was surprised to find that the foramen of Winslow just admitted my forefinger with difficulty.

Subsequent Progress.—The baby had an uneventful recovery, and was discharged from hospital at the end of two weeks.

Discussion.—This type of case is rare at all ages. Sir Lancelot Barrington-Ward[1] cites only 34 reported cases. He quotes Haw, who described the case of a

coloured boy 5 years of age who died 2½ hours after the onset of acute obstruction. At the post-mortem examination the cæcum, appendix, and part of the ileum lay behind the stomach, having passed through the foramen of Winslow. Reduction was impossible until the gastro-hepatic omentum had been divided.

H. S. Souttar[2] says that, "internal hernias are fortunately rare, since if strangulation occurs it is almost invariably fatal. Reduction is difficult and often impossible".

In my case I was fortunate to have been able to operate within an hour of the acute obstruction, and I am now convinced that complete reduction was possible owing to the fact that the last half-inch of the long appendix had not disappeared through the foramen.

Looking back on this case, there is one point of self-criticism I would make. Although there was no congenital deformity of the intestinal attachment, the area of intestine affected was exceedingly lax in its mesenteric attachment, and I ought, perhaps, to have risked a few more minutes in the fixation of the ascending colon. Volvulus of the ileocæcal segment is rare in the child, but, since herniation was possible, I wonder now if another accident is possible in later life.

REFERENCES

[1] BARRINGTON-WARD, SIR LANCELOT, *The Abdominal Surgery of Children*, 216. London: Oxford University Press.
[2] SOUTTAR, H. S., *The Art of Surgery*, 472. London: Heinemann.

REVIEWS AND NOTICES OF BOOKS

Diseases of the Thyroid, Parathyroids, and Thymus. By ANDRÉ CROTTI, M.D., F.A.C.S., LL.D., R.I.C.S. Third edition, revised and enlarged. 10½ × 7¼ in. Pp. 1229, with 262 illustrations and 39 coloured plates. 1938. London: Henry Kimpton. 90s. net.

DR. CROTTI's book, originally entitled *Thyroid and Thymus*, is now republished in a third edition as *Thyroid, Parathyroid, and Thymus*. It has increased in size from 774 to 1229 pages, its weight has gone up from nearly 7 lb. to 9 lb., while the illustrations are almost trebled. So massive a tome is difficult to handle, though the publishers have done their work well and the book is beautifully and clearly printed and bound, even if the paper is unnecessarily thick.

The reviewer, familiar with previous editions, has been at some pains to examine in detail the additions and changes in this, the third.

There is a chapter of nearly 100 pages giving details of the author's work on the aetiology of endemic goitre; a fuller treatment of thyroid insufficiency and cretinism; and a greatly enlarged and more accurate index, which might have been improved, however, by the use of graded type. Most of the other chapters have been amplified, some of them greatly so, and while on the whole this is to the good, the information is too often supplied in a categoric fashion without that evidence of critical discrimination which we are entitled to expect in such a work. Dr. Crotti is apparently better acquainted with the German, French, and American medical literature than with the English, and this has led him into a number of errors which might easily have been avoided. He says, for instance (p. 23), that aberrant thyroid glands have a normal histological structure, whereas Dunhill has shown that as a rule they possess a papilliferous structure. He also describes the thyroid at the end of intra-uterine life as being a solid undifferentiated mass of cells without alveoli, though Murray has clearly demonstrated the presence of well-formed alveoli containing colloid at birth. The author gives a very full account of recent advances in our knowledge of thyroid physiology, but makes the curious statement that thyroxine was synthesized in 1919, though Barger and Harington's great achievement dates back only to 1926.

A good description follows of the relationship between the thyroid and other endocrine glands, and particularly of the interaction of pituitary and thyroid in connexion with the aetiology of thyrotoxicosis. He emphasizes the valuable point that the effect of the pituitary thyrotropic hormone is temporary, and that the thyroid soon enters into a refractory stage.

Few would disagree with the author in condemning Crile's operation on the adrenal sympathetic nerves for thyrotoxic conditions, since the adrenal is not primarily at fault, and the operative risk is immeasurably greater than in thyroidectomy. It seems hardly necessary, however, to have included eighteen pages of description of the sympathetic system, as this information is better given in special monographs.

The author is at the best in describing the clinical features of thyroid disease, particularly in the chapter on malignant goitre. He unfortunately does not adopt A. Graham's simplified classification, but adheres closely to that of Langhans, from which such dubious types as parastruma might with advantage have been eliminated. He also appears to be unaware of Trotter's convincing explanation of the so-called 'cancroid' tumours as being really secondary to post-cricoid hypopharyngeal epitheliomata. When, however, Dr. Crotti says that any hard nodule on the thyroid should be viewed with suspicion with regard to malignancy, this is sage advice which all interested in thyroid diagnostics should take to heart. The author devotes eight pages to the aetiology of malignant disease in general, and here again it seems that this is a subject out of place in a book of this scope.

The chapter on inflammation of the thyroid is one of the best, though Kocher's view that acute inflammation occurs only in goitrous glands is not borne out by those whose experience is not confined to goitrous districts. Under 'woody thyroid' the author describes eight cases from his own practice, none of which justify the ascription, for Dr. Crotti was able to carry

out wedge resections of the lateral lobes, a sheer impossibility in true Riedel's disease, though relatively easy in Hashimoto's lymphogranuloma, which his cases closely resemble. It is a pity that only twelve lines could be spared for hydatids of the thyroid, as, though rare, a fuller account might have been expected in such a lengthy monograph.

In describing the pressure effects of goitre, the author's experience is that the diffuse simple goitre rarely causes tracheal compression, which contrasts with Berry's observation in this country that bilateral tracheal compression is found in its most dangerous form in the colloid goitre of adolescence. Crotti wisely says that in severe suffocative goitre thyroidectomy is always preferable to tracheotomy. Total loss of voice is said to follow bilateral compression of the recurrent laryngeal nerves, though, in fact, complete aphonia in thyroid disease is invariably functional. The book contains an excellent summary of the Kocher-Reverdin controversy as to priority in recognizing cachexia strumipriva. The chapters relating to hypothyroidism and cretinism are among the most interesting, though cautious clinicians would hardly admit the existence of the numerous types of masked hypothyroidism he describes, nor would many to-day agree that hypothyroidism is sometimes associated with a mobile cæcum and curable by cæcopexy!

Dr. Crotti lapses again into irrelevancy by discussing at some length pituitary, water-salt, and genital obesity, but atones by following this up with a thoughtful and well-balanced account of the aetiology of endemic goitre, in which he states fairly the case for and against the iodine-deficiency theory, though he omits to quote the most damning evidence against the theory provided by Hercus, Benson, and Carter in their later work in New Zealand. It is perhaps natural that he should state the evidence for his own theory, i.e., that endemic goitre is due to a yeast-like fungus, but surely one hundred pages of it even in this monster volume is excessive. There is practically no confirmation of the facts alleged, and the flaws in the evidence are only too evident.

The part of the book which in former editions was devoted to Graves' disease is now placed under thyrotoxicosis, which facilitates the description of all varieties of thyroid toxæmia under one heading. With most modern authorities he regards as invalid Plummer's rigid differentiation of thyrotoxicosis into two types. Regrettably he fails to do justice to Parry's undeniable claim to priority in describing both primary and secondary thyrotoxicosis (though he did not use such terms).

Dr. Crotti again overlooks the work of English pathologists when he ignores the fact that it was Greenfield in 1893 who first recognized the hyperplastic type of structure in Graves' disease, since he gives the credit to Lubarsch (1896).

There is little help provided in the book in differentiating thyrotoxicosis from 'neuro-circulatory asthenia', but Dr. Crotti asserts that the great majority of shell-shock, irritable heart, and so forth were developed on a thyrotoxic basis, whereas Sir Thomas Lewis's conclusions are diametrically opposite. There is much which is good under the heading "masked hyperthyroidism", but the author unnecessarily complicates an already sufficiently obscure subject by demarcating various subtypes which are rarely if ever identifiable in practice. He explains the pathology of masked hyperthyroidism as due to hypertrophy and hyperplasia of the interstitial and fœtal cells, yet the most reliable authorities, such as Rienhoff, could find no trace of such cells in the normal gland.

Crotti gives a sane account of the pros and cons of medical, radiological, and surgical treatment in thyrotoxicosis, but he adds little to what is already well known. He relegates X-ray therapy to the incipient and to the bad-risk cases. His operative technique is similar to that in the best clinics in this country, though he has failed to appreciate the vital necessity for ligation of the inferior thyroid arteries if recurrent thyrotoxicosis is to be reduced to a minimum. Crotti does not employ preliminary ligation to any great extent, nor does he do thyroidectomy in stages, implying that as a rapid operator he sees no object in taking two bites at a cherry. He seldom drains the wound, a practice which is undoubtedly growing elsewhere.

The book ends with a long chapter on the thymus and its diseases, which the reviewer found tedious to follow as so little of the subject matter appears to be based on personal experience, unlike the chapters on the thyroid, which are vividly written. Dr. Crotti is one of the diehards who ignore all evidence against the view that the thymus has an influence on thyrotoxicosis, and removes as much as possible of the thymus whenever it is obviously hyperplastic in thyrotoxic cases. The Mayos and Lahey in America, to say nothing of English and Continental surgeons, have exposed the fallacy of this procedure.

It would be pleasant to be able to say that in this edition the numerous typographical and other errors of detail had been corrected. Unfortunately the same slovenliness is exhibited as in the last edition. As an example, on p. 891, in six lines there are three errors which have not been corrected from the last edition. Blizard is spelt Blizzard, Walther is spelt Walter, and Porta as Porter. Elsewhere Dalrymple becomes Dallrymple, Goodall—Goodal, Leitch—Leibet, etc., etc. Your reviewer failed to trace in the Bibliographic Index many of the names cited in the text, but after counting a hundred such omissions abandoned the task. It is to be hoped that in future editions Dr. Crotti will add the titles of the papers cited in the Bibliographic Index and correct the irritating errors in the text. It does not seem unreasonable to expect a high standard of accuracy in a book which is an expensive one even by American standards.

Das Kropsproblem. By Dr. EUGEN BIRCHER, Aarau, Switzerland. 8vo. Pp. 143, with 41 illustrations. 1937. Dresden and Leipzig: Theodor Steinkopff. RM. 12; bound RM. 13.

THIS is one of a series on practical medicine for post-graduates. As such, it deals with the problems of goitres, chiefly from the practical points of view—namely, those of diagnosis and treatment. Anatomy, physiology, and pathology are briefly described, and the bulk of the book consists in the diagnosis and treatment of various types of thyroid anomaly. Illustrations consist for the most part of photographs of patients sufferings from goitre. The operative treatment is very shortly described without any illustrations.

The Hypothalamus: Morphological, Functional, Clinical and Surgical Aspects. By W. E. LE GROS CLARK, D.Sc., F.R.C.S., F.R.S.; JOHN BEATTIE, M.D., D.Sc.; GEORGE RIDDOCH, M.D., F.R.C.P.; and NORMAN M. DOTT, F.R.C.S. Edin. 10 × 7½ in. Pp. 212 + xii, with over 100 illustrations. Published for The William Ramsay Henderson Trust. Edinburgh: Oliver & Boyd. 12s. 6d. net.

WHEN seeking his asses Saul found a kingdom, and folly is often so closely allied to what is of much value, that Gall and Spurzheim were not only the founders of phrenology but also gave rise to the important doctrine of cortical localization. When Mr. William Ramsay Henderson died in Edinburgh in 1832 he provided a sum of money for "the advancement and diffusion of the Science of Phrenology". He has, however, sown better than he realized, for the fund has made possible the series of lectures on the hypothalamus which were delivered in Edinburgh in 1936, and now appear in amplified form as this book. The authors would be the last to claim that it is a complete treatise on the hypothalamus, but the writer of each of the four sections of which it is comprised summarizes our knowledge of the aspect with which he deals: Le Gros Clark, the morphological; John Beattie, the functional; George Riddoch, the clinical; and N. M. Dott, the surgical. When asked by a student where the sympathetic nervous system began, Sir William Turner is said to have nearly frightened him out of his wits by roaring at him, "The sympathetic system, sir, has no beginning". To-day the reply might have been "in the hypothalamus", but this region, which forms that part of the floor of the brain cavity in immediate topographical relation with the pituitary body, and which is phylogenetically speaking one of the most ancient parts of the vertebrate forebrain, has within recent times had a multitude of functions ascribed to it. There can be little doubt that it constitutes part of a mechanism concerned with the more primitive emotional reactions such as laughter and tears, the sexual functions, and with sleep and waking. Harvey Cushing, however, has reminded us that there has been a tendency to overload the thalamus with functional responsibilities, and the balanced account of our present knowledge of this fascinating region which is set forth in this monograph will act as a salutory corrective to any such attempt. Some few years back Cushing himself aroused much comment when he suggested an association between certain hypothalamic lesions and gastro-duodenal ulceration, but it is interesting to note that Norman Dott has some convincing records of such an association in at least two of the cases which he reports here. The account of the surgical aspects will appeal particularly to surgeons, and the masterly way in which, by a double approach from above and below, Dott has been able to attack a series of hypothalamic tumours with cystic extension into the third ventricle makes interesting reading.

There are only two illustrations in the clinical section, and this is regrettable owing to their excellence throughout and to the lavish distribution in the other sections. The type is large and clear, and the matter particularly well arranged. Physiologists, neurologists, and neurological surgeons wherever they may be will want to add this work to their libraries, and its appeal should be wide.

The Pituitary Gland. An Investigation of the most Recent Advances. The Proceedings of the Association for Research in Nervous and Mental Disease, New York, December 28 and 29, 1936. Editorial Board: W. TIMME, A. M. FRANTZ, C. C. HARE. 9 × 6 in. Pp. 764 + xxiv, with 160 illustrations and 53 tables. 1938. Baltimore: The Williams Wilkins Co. (London: Baillière, Tindall & Cox). 45s. net.

THIS book is volume xvii of a series of publications on behalf of the Association for Research in Nervous and Mental Disease. Over forty contributors have taken part in its compilation, and every aspect of the pituitary gland in health and disease has been studied. The book itself is divided into three sections. The first section is devoted to the anatomy of the gland, and the chapter dealing with the cytology of the pituitary is very comprehensive and extensively illustrated. The second section deals with the physiology of the gland, and covers nearly three hundred pages. The different hormones and anti-hormones are discussed in detail; while the relation of the anterior pituitary gland to problems of puberty and menstruation are considered in full. Such questions as the pituitary gland in relation to water exchange and the influence of light upon pituitary activity are studied *in extenso*.

The third section is of greater interest to surgeons, for it covers the clinical manifestations and surgical treatment of pituitary tumours.

The book will remain a reference volume for many years to come, because all that is known regarding this gland has been incorporated in it. The illustrations are excellent and are a real feature of the book.

A Text-book of Surgical Pathology. By C. F. W. ILLINGWORTH, M.D., F.R.C.S. (Edin.), Lecturer in Clinical Surgery, University of Edinburgh; Senior Assistant Surgeon, Edinburgh Western Hospital; and B. M. DICK, M.B., F.R.C.S. (Edin.), Lecturer in Clinical Surgery, University of Edinburgh; Assistant Surgeon, Edinburgh Western Hospital. Third edition. 10 × 6¼ in. Pp. 727 + viii, with 299 illustrations. 1938. London: J. & A. Churchill Ltd. 36s. net.

THOSE who read this book will understand why the demand has necessitated the publication of three editions within the space of six years. By a process of careful deletion the authors have succeeded in keeping the third edition about the same size as the first and second, although important revisions and additions have been made. The latter include excellent descriptions of obliterative diseases of the blood-vessels, tumours of nerves, fat embolism, and metatarsal dystrophies. In these dystrophies are included metatarsalgia, marching foot, and Köhler-Freiberg disease of the metatarsal heads. Chapters devoted to the thyroid, pituitary, and adrenal glands have been brought up to date.

It would be difficult in this excellent treatise to find any one chapter or portion which excels another, and a careful search fails to find anything which would justify adverse criticism. There are perhaps a few omissions, such as disorders of the sympathetic nervous system, for which room may be found in future editions. The sections on gas gangrene, surgical shock, the origin and irradiation of tumours, the various diseases of bone, and the surgical pathology of the genito-urinary tract, will appeal to those who are devoted to the science as well as the art of surgery.

The book overflows with practical information, linking clinical with pathological facts, and bedside problems are clarified by reference to its contents.

A Short Practice of Surgery. By HAMILTON BAILEY, F.R.C.S. (Eng.), Surgeon, Royal Northern Hospital, London; Surgeon and Urologist, Essex County Council; Surgeon, Italian Hospital., etc.; and R. J. MCNEILL LOVE, Surgeon, Royal Northern and Metropolitan Hospitals; etc. Fourth edition. 8½ × 5½ in. Pp. 996 + viii, with 818 illustrations, of which 109 are coloured. 1938. London: H. K. Lewis & Co. Ltd. 28s. net.

EXAMINERS all over the country have often been impressed by the popularity of this text-book amongst students of medicine.

In reviewing the new edition, the reason for this is clearly seen. The authors present the subjects dogmatically, the illustrations (many of them in colour) are excellent, and the information supplied is always neatly classified and tabulated. These characteristics impress the student. He is often able to repeat verbatim the exact words of Bailey and Love in an examination paper. Surgeons may disagree as to the advisability of this kind of knowledge, but students are conservative and have always preferred the more dogmatic type of text-book.

The new feature of footnotes in italics giving short explanatory notes of proper names perhaps exemplifies the extreme pruning of the authors. The footnote under page 788, for example, reads: "Baron Guillaume Dupuytren, 1777–1835. Surgeon Hôtel Dieu, Paris. Very parsimonious, and died a millionaire". Interesting no doubt, but an extreme case of blue pencilling. If anything of this kind is contemplated in a new edition, the reviewer would suggest something more on the lines of the biographical index of Homan's text-book.

Industrial Surgery: Principles, Problems, and Practice. By WILLIS W. LASHER, M.D., F.A.C.S., Assistant Professor of Traumatic Surgery, New York Post-Graduate Medical School; etc. 9½ × 6¼ in. Pp. 452 + xxv, with 193 illustrations. 1938. New York: Paul B. Hoeber Inc. $6.00.

THE author has attempted in this small volume to describe the scope of industrial surgery as practised in the U.S.A. The two chapters devoted to the organization and equipment of an industrial clinic are of considerable interest. The remainder of the book is, however, disappointing. The description of the clinical features and treatment of the various injuries is far too brief, and in any case inaccurate. Many of the methods advocated would not meet with the approval of British surgeons experienced in this field of surgery.

The disability percentages given at the end of each chapter will be useful to those engaged in industrial practice in the United States, but can be of little value to surgeons elsewhere.

The Practice of Urology. By LEON HERMAN, B.S., M.D., Professor of Urology, University of Pennsylvania Graduate School of Medicine; Urologist to the Pennsylvania Hospital and to the Bryn Mawr Hospital; etc. 9½ × 6¼ in. Pp. 923, with 504 illustrations. 1938. Philadelphia and London: W. B. Saunders Co. Ltd. 45s. net.

DR. LEON HERMAN is Professor of Urology in the University of Pennsylvania, and, judging by the outstanding merit of this book, is hardly as well known on this side of the Atlantic as he should be. Undoubtedly he is deeply versed in his subject, and he appears to have read nearly everything pertaining to genito-urinary surgery; yet he refuses to allow himself to be submerged by the opinions of others, and the personal note is predominant throughout.

The volume has been admirably produced, and, with the exception of a few of the photographs, the illustrations are beautiful; they are a credit to the artist and the publishers, and will be a source of pleasure to many surgeons and urologists. These and the X-ray films have been selected with care, and the accompanying legends are often as interesting and as instructive as the text.

Each chapter is followed by an excellent bibliography, all the more useful for not being too copious. We observe with interest that the authors he quotes are mostly American, though he mentions Bland-Sutton, Thomson-Walker, and Swift Joly. Some French and German authors are mentioned, but we venture to congratulate Dr. Leon Herman on his escape from an obsession from which some of his colleagues appear to suffer—that urological wisdom is to be sought only in a part of the continent of Europe.

In a treatise so up to date and so sound in doctrine it is difficult, and it would be ungrateful, to criticize, but our readers may be interested to learn the author's views on some of the more unsettled questions of treatment.

On the still vexed question of urinary antiseptics he gives ample, but somewhat sceptical, information; he believes that they may control, but rarely destroy, chronic urinary infections, and he states that mercurochrome is neither useful nor safe. He writes a full account of prontosil, but thinks it is too early to assess its real value.

He favours the use of gonadotropic hormones in the treatment of retention of the testis, but seems to view the results without enthusiasm; on the other hand, the hormonal test (Aschheim-Zondek) is considered a valuable aid in the differential diagnosis of intrascrotal tumours and, perhaps even more so, in their prognosis.

Of the operative treatment of prostatism he says that two-thirds of his own cases are treated by transurethral resection; it is his belief that a prostatic tumour of such size as to need two or more resections for its removal should be enucleated.

The sections on carcinoma of the prostate, in which he condemns the use of radium, on injuries of the ureter, and on congenital abnormalities of the kidney, are amongst the best in the book, and perhaps as good as any that have been written on these subjects. There is a fine example, with an illustration, of a retrocaval ureter with dilatation of the proximal part.

If one of the criteria of a good book on surgery is that it will be found valuable for reference in case of difficulty or doubt, then we believe that this reaches that standard, for it is full of curious and unusual cases.

When a second edition is called for, we venture to call the attention of Dr. Leon Herman to the existence of what is sometimes called Schramm's phenomenon and sometimes funnel-shaped deformity of the bladder neck. A description of this can have been omitted only by inadvertence, for we should hesitate to believe that a writer of such industry and erudition does not know all about this curious condition.

We have no hesitation in heartily commending this work to our readers; we are sure that it will become a classic and will find a place on the shelves of every urologist and of many a surgeon. We congratulate the author and his university on a notable accomplishment.

Wundversorgung und Wundbehandlung. By Prof. Dr. HANS VON SEEMEN, Munich. Heft 19 of *Vorträge aus der praktischen Chirurgie* (Lexer). Large royal 8vo. Pp. 66. 1938. Stuttgart: Ferdinand Enke. Paper covers, RM. 4.60.

THIS is another monograph in the practical surgery series initiated and edited by Professor Eric Lexer. It opens with a portrait and memorial notice of Prof. Lexer, who died suddenly in 1937.

It deals with the care and treatment of different types of wounds. After a short description of the process of wound healing, it discusses the details of wound toilette. Very much more importance is attached to mechanical than to chemical disinfection, but both alcohol and iodine are freely used. A detailed discussion is given of wound drainage, different types of wound infection, and the methods of dealing with foreign bodies. The use of the electric cautery or diathermy is considered, especially in the matter of primary wound excision. Finally, there is a short discussion as to the metabolic changes which accompany severe trauma.

Die Kriegsschussverletzungen der Gelenke und Knochen der Gliedmassen. By Prof. D. KARL FRANZ. Heft 18 of *Vorträge aus der praktischen Chirurgie* (Lexer). Large royal 8vo. Pp. 30. 1938. Stuttgart: Ferdinand Enke. Paper covers, RM. 1.80.

THE treatment of gunshot wounds of the joints and bones is very briefly described, and each section is followed by some statistical information as to the mortality and the recovery of these injuries in the late war in the French, American, and German armies.

Akute Gliedmassendystrophie in ihrer Bedeutung für die Behandlungsmassnahmen in der Unfallchirurgie. By B. KARITZKY, Freiburg. 9¼ × 6¼. Pp. 52. 1938. Berlin: F. C. W. Vogel. RM. 4.40.

THIS and the following monograph are part of the monthly journal for traumatic surgery (*Unfallheilkunde*). Both these monographs deal with the condition of atrophy or distrophy which follows injuries to the limb and which is often known as Sudeck's atrophy, after the name of Dr. Paul Sudeck, who first described it in 1900. The most characteristic feature of the condition is the mottled decalcification of the bones; with this are associated shiny atrophic skin, wasted muscles, œdema, and finally ankylosis of the joints. This condition may follow injury, any infective inflammation, or nerve lesion. For the most part this paper is devoted to considering how the condition may be avoided. In the treatment of the skin prolonged preparation by washing, etc., is advised. A closed plaster dressing is the best after-treatment for wounds, but the use of plaster-of-Paris without padding is considered to be dangerous. The initiation of active movements after the limb has been put up in plaster is of the utmost importance. A very strong plea is made for the early operative treatment of those limb injuries in which there is gross bone displacement or rupture of ligaments or tendons.

Kollaterale Entzündungszustände (sog. akute Knochenatrophie und Dystrophie der Gliedmassen) in der Unfallheilkunde. By Dr. PAUL SUDECK. Heft 24 of *Monatsschrift für Unfallheilkunde und Versicherungsmedizin* (zur Verth). 9½ × 6¼ in. Pp. 68. with 44 illustrations. 1938. Berlin: F. C. W. Vogel. RM. 6.40.

DR. PAUL SUDECK writes on the same subject in the same series, but he calls it a "collateral inflammatory condition". His monograph is of great value, because it represents the long experience of the surgeon who first described this condition. He distinguishes between three stages of this condition—namely: (1) The stage of inflammation; (2) The stage of dystrophy (chronic inflammation); (3) The stage of atrophy. Six examples are given and illustrated of each of these stages.

It has been suggested that this condition is due to vasomotor disturbance and that sympathetic operations will relieve it. Sudeck points out that such treatment would be worse than useless in the first or acute stage, but that it may give brilliant results in the second or dystrophic stage when the limb is cold and cyanosed.

Die Arthroskopie (Endoskopie des Kniegelenks). By Dr. med. ERNST VAUBEL, Frankfurt-a.-M. Bd. 9 of *Der Rheumatismus* (R. Jürgens). 9⅝ × 5¾ in. Pp. 66 + viii, with 25 illustrations, 16 of which are in colour. 1938. Dresden and Leipzig: Theodor Steinkopff. RM. 10.

THIS is one of a number of monographs contributed by various authors to the elucidation of various joint problems called by the general name of rheumatism. The knee-joint is examined under a local anæsthetic and filled with fluid or air, and the interior examined by means of a straight endoscope. There are appended twenty-five beautifully coloured drawings, showing the various conditions found in the knee-joint in various inflammatory conditions. There can be no doubt that with sufficient practice this method would be most useful in the diagnosis of various conditions of internal derangement of the knee.

Diabetes und Chirurgie. By Prof. Dr. A. W. FISCHER, Direktor der Chirurgischen Universitäts-Klinik, Giessen. 8vo. Pp. 44. 1937. Stuttgart: Ferdinand Enke. RM. 2.

THIS is one of a series of monographs dealing with practical surgery. After a short description of the characters of pancreatic diabetes, the diagnosis and estimation of the severity of the disease is discussed. Diabetes is then considered as a complicating factor in various surgical conditions, and it is urged that where life-saving operations are required these should be as simple as possible, e.g., drainage of the gall-bladder rather than cholecystectomy should be performed in cases of gall-bladder disease. Two conditions which owe their origin often to diabetes—namely, carbuncle and gangrene—are described in some detail. The type of amputation required in the latter will depend largely upon the coexistence of arteriosclerosis. The general dietetic and insulin treatment of diabetes is discussed, together with the influence of trauma upon the disease. Finally, a short description is given of experiments for the operative treatment of diabetes. These consist in the denervation of the adrenal gland.

Die Therapie der Thrombose. By Dr. ERNST FRIEDLÄNDER. 9⅜ × 6 in. Pp. 117, with 27 illustrations. 1938. Leipzig and Vienna: Franz Deuticke. RM. 5.40; bound RM. 6.00.

THIS small monograph is almost entirely concerned with the problems of venous thrombosis, its origin, prevention, and treatment; this includes a description of varicose veins and their treatment by pressure and injections. Very great importance is attached to various statical conditions, and a large proportion of the little line drawings depict the various attitudes of the patients lying, reclining, or sitting in bed. It is suggested that the matter is of sufficient importance to justify the provision of special hospitals or departments for dealing with this subject.

The Surgical Treatment of Hypertension. By GEORGE CRILE, edited by AMY ROWLAND. 9½ × 6 in. Pp. 239, with 52 illustrations. 1938. Philadelphia and London: W. B. Saunders Co. Ltd. 18s. net.

DR. CRILE believes "that in individuals who are subject to hypertension, the cœliac ganglia, the cœliac plexus, and the aortic plexus, by inheritance, are larger than in normal individuals.

That excision of the cœliac ganglia, breaking of the cœliac plexus and aortic plexus, resection of all the splanchnic nerves, and denervation of the adrenal gland in order to lessen the drive of the sympathetic ganglia in the adrenal medulla, would affect essential hypertension if it is still in its physiologic phase. . . ." He has reached this view after a study of the morphology of the sympathetic system in a large number of animals. The first part of his book contains a disconnected account of the study, and an attempt to fit his hypothesis to the disturbed control of circulation and oxidation, which he terms the " pathologic physiology of the mechanism ", and which he identifies as the cause of essential hypertension. Thereafter three of his colleagues contribute well-written chapters on certain medical aspects of hypertension. Dr. Crile then describes the technique of the operation which he recommends at present; this is cœliac ganglionectomy and denervation of the aortic plexus. It is carried out through a lumbar incision on both sides, and entirely by touch. Those who are familiar with Dr. Crile's surgical dexterity may possibly accept the blind method in his hands; but it does not commend itself in an age when the keynote of operative surgery is adequate exposure.

The results of this operation in 85 cases are given in a series of tables. Sixteen patients have been followed for more than a year, and of these 12·5 per cent are reported as having " completely normal " pressures. In the chapter devoted to illustrative case histories 6 cases are reported: in 3 of these the operation was unilateral. A brief summary of the work of others in this field completes the book.

The material is presented in a style which is not usually associated with a serious monograph, and the book will not appeal to those who seek in its pages a critical review of the whole subject. However, a declaration of faith by Dr. Crile is always stimulating, although the reviewer feels that his contentions might have been supported by more complete clinical information.

Actinomycosis. By ZACHARY COPE, B.A., M.D., M.S. (Lond.), F.R.C.S. (Eng.), Surgeon to St. Mary's Hospital, Paddington; Senior Surgeon to the Bolingbroke Hospital, Wandsworth Common; etc. $8\frac{3}{4} \times 5\frac{5}{8}$ in. Pp. 248 + xii, with 51 illustrations and 8 coloured plates. 1938. London: Oxford University Press Ltd. 15s. net.

THIS is a monograph of 240 pages of comfortably large print containing many pictures excellently produced. It really would seem to be a complete presentation of all the knowledge of this disease which has accrued to date. It will undoubtedly become the standard work of reference from which any future investigator will set out. The list of authorities which the author has consulted occupies twenty-six pages; he cannot have overlooked much. The outcome of his researches is a very pleasant account of actinomycosis in all its phases: and it is a valuable account, very helpful in diagnosis, an aid to prognosis, and sound on treatment. The author does not believe in the very large doses of potassium iodide recommended by some clinicians, as he finds their claims unsubstantiated; and he preserves an admirably balanced judgement in discussing the use of radiation and the less common forms of treatment.

Although there is nothing particularly new in the book, we doubt if any surgeon could read it through without coming upon facts hitherto unknown to him.

Gray's Anatomy, Descriptive and Applied. Edited by T. B. JOHNSTON, M.D., Professor of Anatomy in Guy's Hospital Medical School, University of London; and J. WHILLIS, M.D., M.S., Reader in Anatomy, Guy's Hospital Medical School, University of London. Twenty-seventh edition. 10 × $6\frac{1}{2}$ in. Pp. 1536 + xxxii, with 1336 illustrations, of which 624 are coloured and 29 are X-ray plates. 1938. London: Longmans, Green & Co. Ltd. 45s. net.

THERE can be very few books which have so faithfully served so many generations of medical students as *Gray's Anatomy*. It was written by Henry Gray (1827–61) of St. George's Hospital when he was just over 30, and the first edition of 750 pages and 863 figures appeared in 1856. It is remarkable that although this text-book has had many rivals it still holds its place as the most widely popular text-book of anatomy. This no doubt is due to the skill and pains taken by the successive editors, Timothy Holmes, Pickering Pick, Howden, and lastly, since 1930, Professor T. B. Johnston.

In the present edition two sections have been largely re-written. The section on joints has had incorporated in it a detailed discussion of movements, and particularly unusual accessory

movements, a knowledge of which is so important in manipulative surgery. That on the lymphatics has been rewritten by Dr. Whillis, who has described in detail the areas of lymphatic drainage. The section on the brain which now forms such a large part of modern anatomical teaching, contains a new description of the connections of the thalamus and the nuclei of the hypothalamus. A welcome innovation is the introduction of X-ray photographs, the understanding of which should now form a part of every student's teaching whilst he is in the dissecting-room.

Some of the older figures have been discarded and nearly 100 new illustrations have been introduced in their place. Many of these new figures are by Mr. A. K. Maxwell, who stands pre-eminent as an anatomical artist. Curiously one of the new figures has introduced a serious error. This is *Fig.* 560, showing a section through the knee-joint. In this the quadriceps tendon is shown as ending in the upper border of the patella and the ligamentum patellæ as arising from the lower and posterior border of the patella, and the continuity of the quadriceps tendon over the front of the patella is not shown. This point, which has such an important bearing on the treatment of transverse fractures of the patella, was quite correctly delineated in the twenty-fifth edition.

We envy the lot of the modern student in having such a beautiful work of science and art at his disposal.

Celsus : De Medicina. With an English Translation by W. G. SPENCER, M.S. (Lond.), F.R.C.S. (Eng.). In three volumes (Loeb Classical Library). $6\frac{5}{8} \times 4\frac{1}{4}$ in. Vol. I (1935). Pp. 499 + xiv. Vol. II (1938). Pp. 292 + lxviii. Vol. III (1938). Pp. 293-649 + vi. London : William Heinemann Ltd. Each volume, cloth, 10s. ; leather, 12s. 6d.

WAS Celsus a medical man ? This question has been debated warmly by scholars. They do not agree. Many hold the view that he was a professional writer, an encyclopædist, who copied and borrowed from the works of Hippocrates, Asclepiades, and other medical authors. Seeing that experts disagree, the humble clinician may perhaps be allowed to express the opinion that it would be difficult, almost impossible, for a layman to write such a book, with such a mass of technical detail. It would seem that if Celsus did not possess the registrable qualification of the time, he had at any rate a sound knowledge of medicine, and we may leave it at that.

Practically nothing is known of the life of the man, though from internal evidence it is surmised that he was born about 35 B.C., and that he lived in Rome during the reign of the Emperor Tiberius.

His contemporaries do not seem to have thought much of him, for Quintilian (C. A.D. 35–95) dubs him " a man of mediocre ability ". But Celsus has his revenge, for his work was the first medical book to be printed in 1478, and since then there have been innumerable editions and translations, and it has been used as a text-book in England until comparatively recent times.

The treatise " On Medicine " is divided into eight books. It commences with an introduction, the Proœmium, which contains a sketch of the history of medicine from the earliest times and ends with the sound conclusion that the art of medicine should be based on reason and a good knowledge of anatomy. The first two books deal mainly with considerations of diet and régimen, and are interesting to us, now, as illustrating the thought of the time. Book III discusses internal diseases, and in it comes the time-honoured description of the signs of inflammation which have been hammered into the heads of countless students :—

> " Now the signs of inflammation are four ;
> Redness and swelling with heat and pain ".

Books IV and V deal with external diseases and medicaments, of which there is a formidable list. The portion dealing with the treatment of wounds is interesting, and incidentally one finds here the first account of ligature of vessels : " . . . the blood-vessels which are pouring out blood are to be seized, and round the wounded spot they are to be tied in two places and cut across between " (Book V, 26).

For the surgeon Books VII and VIII are the most interesting. They prove that in the first century A.D. the art of surgery had already attained a considerable degree of perfection. Celsus states that it was more practised by Hippocrates than by his forerunners, and that later " it was separated from the rest of medicine and began to have its own professors ". He also leads us to believe that surgery had progressed in the hands of the Alexandrian surgeons, from whom it had spread to Rome.

The discussion on the opening and treatment of abscesses is clear and rational. The section on the treatment of fistula is well done, and so is that on the removal of missiles. There are directions for the removal of a goitre and for the treatment of hernia. Careful directions are given for the operation for removal of varicocele, and mention is made that the vessels are to be tied with flax thread, the ends of which are to be left hanging out of the wound.

There are many other interesting accounts of operations which are too long to describe here, but sufficient has been said to show that the work is rich in material for the medical historian.

Now a word concerning the translation. This has been done excellently by our octogenarian surgeon-scholar, Mr. Spencer, who by his learning and industry has conferred a benefit on those who do not possess his knowledge of the Classics. What we should like him to undertake now, is to do for Galen what he has done for Celsus.

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.*]

St. Bartholomew's Hospital Reports. Edited by G. EVANS, G. GRAHAM, G. HADFIELD, C. F. HARRIS, W. SHAW, A. WORMALL, Sir G. BALL, J. P. ROSS, and J. MAXWELL. Vol. LXXI. Demy 8vo. Pp. 353 + xxvi. Illustrated. 1938. London: John Murray. 21s. net.

Chirurgie der Lungen und des Brustfelles. By ALFRED BRUNNER, Munich. Bd. 26 of *Medizinische Praxis* (Grote, Fromme, and Warnekros). 8¾ × 6 in. Pp. 282 + xii, with 112 illustrations. 1938. Dresden and Leipzig: Theodor Steinkopff. RM. 22.50; bound, RM. 27.

Modern Anaesthetic Practice. Edited by Sir HUMPHRY ROLLESTON, Bart., G.C.V.O., K.C.B., M.D., F.R.C.P., and ALAN A. MONCRIEFF, M.D., F.R.C.P. 8½ × 5½ in. Pp. 231. 1938. London: Eyre & Spottiswoode (Publishers) Ltd., for *The Practitioner*. 10s. 6d. net.

Everyday Surgery. By LAMBERT ROGERS, M.Sc., F.R.C.S., F.R.C.S.E., F.R.A.C.S., F.A.C.S., Professor of Surgery, University of Wales; Director of the Surgical Unit, Welsh National School of Medicine; Honorary Surgeon, Cardiff Royal Infirmary, etc.; and A. L. D'ABREU, M.B., Ch.M., F.R.C.S., Senior Assistant, Surgical Unit, Welsh National School of Medicine; Assistant Thoracic Surgeon, Welsh National Memorial Association, etc. With an Introduction by Professor G. GREY TURNER. 8⅝ × 5½ in. Pp. 280 + xii, with 160 illustrations. 1938. London: Edward Arnold & Co. 12s. 6d. net.

The Essentials of Modern Surgery. Edited by R. M. HANDFIELD-JONES, M.C., M.S., F.R.C.S., Surgeon to Out-patients, St. Mary's Hospital; Senior Surgeon, Florence Nightingale Hospital, etc.; and A. E. PORRITT, M.A., M.Ch., F.R.C.S., Surgeon to His Majesty's Household; Surgeon to Out-patients and Assistant Director of the Surgical Unit, St. Mary's Hospital, etc. 10 × 6 in. Pp. 1126 + xvi, with 501 illustrations. 1938. Edinburgh: E. & S. Livingstone. 30s. net.

Fractures of the Jaws. By ROBERT H. IVY, M.D., D.D.S., F.A.C.S., Professor of Maxillo-facial Surgery, University of Pennsylvania, etc.; and LAWRENCE CURTIS, A.B., M.D., D.D.S., F.A.C.S., Assistant Professor of Maxillo-facial Surgery, University of Pennsylvania, etc. Second edition, thoroughly revised. 9½ × 6¼ in. Pp. 192, with 199 illustrations. 1938. London: Henry Kimpton. 21s. net.

Chronic Diseases of the Abdomen. A Diagnostic System. By C. JENNINGS MARSHALL, M.S., M.D. (Lond.), F.R.C.S. (Eng.), Surgeon, Charing Cross Hospital and Victoria Hospital for Children; etc. 10 × 6¼ in. Pp. 247 + xviii, with 131 illustrations. 1938. London: Chapman & Hall Ltd. 25s. net.

Röntgendiagnostik der Gelenke mittels Doppelkontrastmethode. By Dr. J. OBERHOLZER. With a Foreword by Dr. med. E. BIRCHER. Vol. 56 of *Fortschritte auf dem Gebiete der Röntgenstrahlen.* 12 × 8½ in. Pp. 128, with 134 illustrations. 1938. Leipzig: Georg Thieme. RM. 23; bound, RM. 25.

Diagnostic Roentgenology. Edited by ROSS GOLDEN, M.D., Professor of Radiology, the College of Physicians and Surgeons, Columbia University, etc. 9½ × 7¼ in. 255 renewal pages containing 230 illustrations. 1938. New York: Thomas Nelson & Sons. $4.50.

Plastic Surgery. By ARTHUR JOSEPH BARSKY, M.D., D.D.S., Associate Surgeon in Charge of the Department of Reconstructive Surgery, Beth Israel Hospital, New York City, etc. 9¼ × 6 in. Pp. 355, with 432 illustrations. 1938. Philadelphia & London: W. B. Saunders Co. 26s. net.

Catalogue of Lewis's Medical and Scientific Lending Library. Part I, Authors and Titles. 8¼ × 5¼ in. Pp. 550 + viii. 1938. London: H. K. Lewis & Co. Ltd. 16s. net (Subscribers, 8s.).

Experience in the Management of Fractures and Dislocations. By the Staff of the Fracture Service, Massachusetts General Hospital, Boston. Under the general editorship of PHILIP D. WILSON, M.D., Surgeon-in-chief, Hospital for Ruptured and Crippled, New York; Clinical Professor of Orthopædic Surgery, College of Physicians and Surgeons, Columbia University, etc. 10½ × 7 in. Pp. 1036 + viii, with 1419 illustrations. 1938. Philadelphia, London, Montreal: J. B. Lippincott Co. 65s. net.

A Manual of Reparative Plastic Surgery. By J. EASTMAN SHEEHAN, M.D., F.A.C.S., Professor of Plastic Reparative Surgery, New York Polyclinic Medical School and Hospital; Surgeon, St. Clare's Hospital, etc. 9¼ × 6 in. Pp. 311 + xxiv, with 314 illustrations and 18 plates. 1938. London: Humphrey Milford, Oxford University Press. 30s. net.

Das Panaritium. By Dr. MAX SAEGESSER, Bern. 9¼ × 6⅛ in. Pp. 97 + iv, with 115 illustrations. 1938. Berlin: Julius Springer. Rm. 4.80.

Medical Research Council Report on Radium Beam Therapy Research, 1934–1937. By CONSTANCE A. P. WOOD, L. G. GRIMMETT, T. A. GREEN, and others, under the direction of the Governing Body of Radium Beam Therapy Research. 9⅝ × 6 in. Pp. 77 + xvi, with 14 figures in the text and 22 plates, some coloured. 1938. London: H.M. Stationery Office. 4s. net.

Modern Surgical Technic. By MAX THOREK, M.D., K.L.H. (France), K.C. (Italy), Professor of Clinical Surgery, Cook County Graduate School of Medicine; Attending Surgeon, Cook County Hospital; Surgeon-in-Chief, The American Hospital; etc. In three volumes. 10¼ × 7 in. Pp. 2045 + xcvi, with 2174 illustrations. 1938. Philadelphia and London: J. B. Lippincott Co. £7 10s. net. Volumes not sold separately.

The Treatment of Fractures. By CHARLES LOCKE SCUDDER, A.B., Ph.B., M.D., F.A.C.S., Consulting Surgeon to the Massachusetts General Hospital; formerly Assistant Professor of Surgery at the Harvard Medical School. Eleventh edition, revised. 9½ × 6¼ in. Pp. 1208, with 1717 illustrations. 1938. Philadelphia and London: W. B. Saunders Co. Ltd. 55s. net.

Technik der Knockenbruchbehandlung. By Dr. LORENZ BÖHLER, Director of the Accident Hospital and Professor of Surgery in the University of Vienna. Sixth edition, in two volumes. Pp. 1532, with 3054 figures. Vienna: Wilhelm Maudrich. RM. 100.